Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
S32	21	ubiquitin SAME (aprataxin OR SLP OR HMG17 OR PinX1 OR CIR OR HMGN3 OR HSPC144)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:48
S31	6	oberoi-p$.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47
S30	4	oberoi-pankaj.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47
S29	58	Biebuyck-H$.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47
S28	97	kenten-j$.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47
S27	2	davydov-I.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47
S6	208	ubiquitin SAME (aprataxin OR tau OR SLP OR HMG17 OR PinX1 OR CIR OR cullin Biebuyck-H$.in. OR HMGN3 OR HSPC144 OR CDC6)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/05/27 15:47

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- Search History will be lost after eight hours of inactivity.
- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

Search	Most Recent Queries	Time	Result
#15	Search **#14 AND #2**	06:32:59	10
#14	Search **(#1 OR #6 OR #8 OR #9 OR #10 OR #11 OR #12)**	06:26:29	5814
#13	Search **(#1 OR #6 OR #8 OR #9 OR #10 OR #11 OR #12) AND #2**	06:26:12	10
#12	Search **"THY-28" [TIAB] OR "THY 28" [TIAB] OR "THY28" [TIAB] OR "HSPC-144" [TIAB] OR "HSPC 144" [TIAB] OR "HSPC144" [TIAB] OR "MDS-012" [TIAB] OR "MDS 012" [TIAB] OR "MDS012" [TIAB] OR "MGC-12187" [TIAB] OR "MGC 12187" [TIAB] OR "MGC12187" [TIAB] OR "MY-105" [TIAB] OR "MY 105" [TIAB] OR "MY105" [TIAB]**	06:25:06	3
#11	Search **"HMGN-3" [TIAB] OR "HMGN 3" [TIAB] OR "HMGN3" [TIAB] OR "TRIP-7" [TIAB] OR "TRIP 7" [TIAB] OR "TRIP7" [TIAB] OR "Thyroid receptor interacting protein 7" [TIAB] OR "Thyroid-receptor-interacting-protein-7" [TIAB] OR "high mobility group nucleosomal binding domain 3" [TIAB] OR "high-mobility-group-nucleosomal-binding-domain-3" [TIAB] OR "PNAS-24" [TIAB] OR "PNAS 24" [TIAB]**	06:24:25	9
#10	Search **"CIR" [TIAB]**	06:19:51	390
#9	Search **"PINX-1" [TIAB] OR "PINX 1" [TIAB] OR "PINX1" [TIAB] OR "PIN2-interacting protein 1" [TIAB] OR "PIN2-interacting-protein-1" [TIAB] OR "PIN2 interacting protein 1" [TIAB] OR "LPTS" [TIAB] OR "MGC-8850" [TIAB] OR "MGC 8850" [TIAB] OR "MGC8850" [TIAB] OR "FLJ-20565" [TIAB] OR "FLJ 20565" [TIAB] OR "FLJ20565" [TIAB] OR "LPTL" [TIAB] OR "TRF1-interacting protein 1" [TIAB] OR "TRF1-interacting-protein-1" [TIAB] OR "TRF1 interacting protein 1" [TIAB] OR "67-11-3 protein" [TIAB] OR "67-11-3-protein" [TIAB] OR "67 11 3**	06:19:00	34

	protein" [TIAB]		
#8	Search **"HMGN-2" [TIAB] OR "HMGN 2" [TIAB] OR "HMGN2" [TIAB] OR "HMG-17" [TIAB] OR "HMG 17" [TIAB] OR "HMG17" [TIAB] OR "MGC-5629" [TIAB] OR "MGC 5629" [TIAB] OR "MGC5629" [TIAB] OR "high-mobility group nucleosomal binding domain 2" [TIAB] OR "high-mobility-group-nucleosomal-binding-domain-2" [TIAB] OR "high mobility group nucleosomal binding domain 2" [TIAB] OR "High-mobility group nucleosome binding domain 2" [TIAB] OR "High-mobility-group-nucleosome-binding-domain-2" [TIAB] OR "High mobility group nucleosome binding domain 2" [TIAB]**	06:18:09	201
#7	Search **"HMG17L1" [TIAB] OR "dJ388M5" [TIAB] OR "dJ388M5.2" [TIAB]**	06:17:36	0
#6	Search **"MLPH" [TIAB] OR "ln" [TIAB] OR "melanophilin" [TIAB] OR "MELPH" [TIAB] OR "MGC-2771" [TIAB] OR "MGC 2771" [TIAB] OR "MGC2771" [TIAB] OR "FLJ-12145" [TIAB] OR "FLJ 12145" [TIAB] OR "FLJ12145" [TIAB] OR "SLAC2-A" [TIAB] OR "SLAC2 A" [TIAB] OR "l1Rk3" [TIAB] OR "l(1)-3Rk" [TIAB] OR "l(1) 3Rk" [TIAB] OR "Slac-2a" [TIAB] OR "Slac 2a" [TIAB] OR "Exophilin 3" [TIAB] OR "Exophilin-3" [TIAB] OR "Synaptotagmin-like protein 2a" [TIAB] OR "Synaptotagmin-like-protein-2a" [TIAB] OR "Synaptotagmin like protein 2a" [TIAB] OR "Slp homologue lacking C2 domains-a" [TIAB] OR "Slp-homologue-lacking-C2-domains-a" [TIAB] OR "Slp homologue lacking C2 domains a" [TIAB] OR "SLAC2A" [TIAB]**	06:16:50	4692
#5	Search **ubiquitin complex AND #1**	06:13:21	0
#4	Search **ubiquitin complex**	06:13:10	4109
#3	Search **#1 AND #2**	06:12:53	0
#2	Search **ubiquitin OR ubiquitination**	06:12:41	13358
#1	Search **"APTX" [TIAB] OR "aprataxin" [TIAB] OR "FLJ-20157" [TIAB] OR "FLJ 20157" [TIAB] OR "FLJ20157" [TIAB] OR "AOA" [TIAB] OR "AOA-1" [TIAB] OR "AOA 1" [TIAB] OR "AOA1" [TIAB] OR "EAOH" [TIAB] OR "MGC-1072" [TIAB] OR "MGC 1072" [TIAB] OR "MGC1072" [TIAB] OR "HGNC:902" [TIAB] OR "FHA-HIT" [TIAB] OR "FHA HIT" [TIAB] OR "AXA-1" [TIAB] OR "AXA 1" [TIAB] OR "AXA1" [TIAB] OR "EOAHA" [TIAB] OR "Forkhead-associated domain histidine-triad like protein" [TIAB] OR "Forkhead-associated-domain-histidine-triad-like-protein" [TIAB] OR "Forkhead**	06:11:51	488

associated domain histidine triad like protein" [TIAB]

Clear History

May 16 2005 17:16:29

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NEWS	5	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	6	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	7	MAR 02	GBFULL: New full-text patent database on STN
NEWS	8	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	9	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	10	MAR 22	KOREAPAT now updated monthly; patent information enhanced
NEWS	11	MAR 22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	12	MAR 22	PATDPASPC - New patent database available
NEWS	13	MAR 22	REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS	14	APR 04	EPFULL enhanced with additional patent information and new fields
NEWS	15	APR 04	EMBASE - Database reloaded and enhanced
NEWS	16	APR 18	New CAS Information Use Policies available online
NEWS	17	APR 25	Patent searching, including current-awareness alerts (SDIs), based on application date in CA/CAplus and USPATFULL/USPAT2 may be affected by a change in filing date for U.S. applications.
NEWS	18	APR 28	Improved searching of U.S. Patent Classifications for U.S. patent records in CA/CAplus
NEWS	19	MAY 23	GBFULL enhanced with patent drawing images
NEWS	20	MAY 23	REGISTRY has been enhanced with source information from CHEMCATS
NEWS	21	MAY 26	STN User Update to be held June 6 and June 7 at the SLA 2005 Annual Conference

NEWS EXPRESS	JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005
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* The CA roles and document type information have been removed from *
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to the file summary sheet on the web at:
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=> e aprataxin/cn
E1            1     APRANAX/CN
E2            1     APRATASTAT/CN
E3            0 --> APRATAXIN/CN
E4            1     APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 2 FRAGMENT)/CN
E5            1     APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 3 FRAGMENT)/CN
E6            1     APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 4 FRAGMENT)/CN
E7            1     APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 5 FRAGMENT)/CN
E8            1     APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 6 FRAGMENT)/CN
E9            1     APRATAXIN (HUMAN GENE APTX 174-AMINO ACID ISOFORM)/CN
E10           1     APRATAXIN (HUMAN GENE APTX 342-AMINO ACID ISOFORM)/CN
E11           1     APRATAXIN (HUMAN GENE APTX SHORT ISOFORM)/CN
E12           1     APRATAXIN (HUMAN GENE APTX SPLICE-VARIANT LE5)/CN

=> s e4
L1            1 "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 2 FRAGMENT)"/CN
```

```
=> d l1

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2005 ACS on STN
RN   625268-59-9  REGISTRY
ED   Entered STN:  09 Dec 2003
CN   Aprataxin (human brain gene APTX isoform 2 fragment) (9CI)  (CA
     INDEX NAME)
OTHER NAMES:
CN   GenBank CAD92455
CN   GenBank CAD92455 (TRANSLATED FROM: GenBank AJ565851)
FS   PROTEIN SEQUENCE
MF   Unspecified
CI   MAN
SR   GenBank
LC   STN Files:   CA, CAPLUS

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=> s e5-e12
             1 "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 3 FRAGMENT)"/CN
             1 "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 4 FRAGMENT)"/CN
             1 "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 5 FRAGMENT)"/CN
             1 "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 6 FRAGMENT)"/CN
             1 "APRATAXIN (HUMAN GENE APTX 174-AMINO ACID ISOFORM)"/CN
             1 "APRATAXIN (HUMAN GENE APTX 342-AMINO ACID ISOFORM)"/CN
             1 "APRATAXIN (HUMAN GENE APTX SHORT ISOFORM)"/CN
             1 "APRATAXIN (HUMAN GENE APTX SPLICE-VARIANT LE5)"/CN
L2           8 ("APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 3 FRAGMENT)"/CN OR
               "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 4 FRAGMENT)"/CN OR
               "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 5 FRAGMENT)"/CN OR
               "APRATAXIN (HUMAN BRAIN GENE APTX ISOFORM 6 FRAGMENT)"/CN OR
               "APRATAXIN (HUMAN GENE APTX 174-AMINO ACID ISOFORM)"/CN OR "APRA
               TAXIN (HUMAN GENE APTX 342-AMINO ACID ISOFORM)"/CN OR "APRATAXIN
                (HUMAN GENE APTX SHORT ISOFORM)"/CN OR "APRATAXIN (HUMAN GENE
               APTX SPLICE-VARIANT LE5)"/CN)

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                                                   ENTRY    SESSION
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=> s l1<CHEM>

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SEL L1 1- CHEM
L3 SEL L1 1- CHEM : 4 TERMS

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FULL ESTIMATED COST	10.93	60.00

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S L3
L4 1 L3

=> s l2<chem>

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FULL ESTIMATED COST	36.03	96.03

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SET COMMAND COMPLETED

SEL L2 1- CHEM

```
L5          SEL L2 1- CHEM :      28 TERMS

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SET COMMAND COMPLETED


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      10.93     106.96

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FILE 'MEDLINE' ENTERED AT 17:39:54 ON 30 MAY 2005

S L5
SEARCH OF L5 IS APPROXIMATELY 71% COMPLETE
   1 FILES SEARCHED...
SEARCH OF L5 IS APPROXIMATELY 71% COMPLETE
   2 FILES SEARCHED...
SEARCH OF L5 IS APPROXIMATELY 71% COMPLETE
L6           4 L5

=> s ubiquit? AND (L4 OR L6)
L7           0 UBIQUIT? AND (L4 OR L6)

=> s ubiquit?
L8      137114 UBIQUIT?

=> s (SLP OR HMG17 OR PinX1 OR CIR or HMGN3 OR HSPC144)
L9         7319 (SLP OR HMG17 OR PINX1 OR CIR OR HMGN3 OR HSPC144)

=> s L8 AND L9
L10         120 L8 AND L9

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11          81 DUP REM L10 (39 DUPLICATES REMOVED)

=> d l11 ibib ti abs 1-81

L11  ANSWER 1 OF 81  CAPLUS  COPYRIGHT 2005 ACS on STN DUPLICATE 1
ACCESSION NUMBER:         2005:156228  CAPLUS
                            Correction of: 2005:16967
DOCUMENT NUMBER:          142:192331
                            Correction of: 142:108390
TITLE:                    Quantitative RT-PCR method for the detection in blood
                          of microarray-identified rheumatoid arthritis-related
                          gene transcripts for diagnosing and monitoring disease
                          state
INVENTOR(S):              Liew, Choong-Chin
PATENT ASSIGNEE(S):       Chondrogene Limited, Can.
SOURCE:                   U.S. Pat. Appl. Publ., 81 pp., Cont.-in-part of U.S.
                          Ser. No. 802,875.
                          CODEN: USXXCO
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DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005003394	A1	20050106	US 2004-812782	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
US 2005003394	A1	20050106	US 2004-812782	20040330
US 2005003394	A1	20050106	US 2004-812782	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812782	A	20040330

TI Quantitative RT-PCR method for the detection in blood of microarray-identified rheumatoid arthritis-related gene transcripts for diagnosing and monitoring disease state

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood for diagnosing and monitoring diseases. The present invention demonstrates that a simple drop of blood may be used to determine the quant. expression of various mRNAs that reflect the health/disease state of the subject through the use of quant. reverse transcription-polymerase chain reaction (QRT-PCR) anal. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring rheumatoid arthritis using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen.

L11 ANSWER 2 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:160724 CAPLUS
DOCUMENT NUMBER: 142:259424
TITLE: Gene expression profiles and biomarkers for the detection of asthma-related and other disease-related gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S. Ser. No. 802,875.

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005042630	A1	20050224	US 2004-816357	20040401
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
US 2005042630	A1	20050224	US 2004-816357	20040401
US 2005042630	A1	20050224	US 2004-816357	20040401
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-816357	A	20040401

TI Gene expression profiles and biomarkers for the detection of asthma-related and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular asthma, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of three records for this document necessitated by the large number of index entries required to fully index the docoment and publication system constraints.].

L11 ANSWER 3 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:9198 CAPLUS
DOCUMENT NUMBER: 142:91478
TITLE: Gene expression profiles in rheumatoid arthritis and osteoarthritis and their use in diagnosis and monitoring disease progress

INVENTOR(S): Blaess, Stefan
PATENT ASSIGNEE(S): Germany
SOURCE: Ger. Offen., 89 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10328033	A1	20050105	DE 2003-10328033	20030619
PRIORITY APPLN. INFO.:			DE 2003-10328033	20030619

TI Gene expression profiles in rheumatoid arthritis and osteoarthritis and their use in diagnosis and monitoring disease progress

AB DNA microarrays that can be used to diagnose and monitor rheumatoid arthritis (RA) and osteoarthritis (OA) are described. Gene expression is analyzed using software that can compare m-dimensional gene expression profiles multi-parametrical with n-dimensional reference gene expression profiles for diagnostics, sub diagnostics classification and therapy decisions.

L11 ANSWER 4 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 2
ACCESSION NUMBER: 2005:492152 SCISEARCH
THE GENUINE ARTICLE: 923DB
TITLE: Misregulation of 2 mu m circle copy number in a SUMO pathway mutant
AUTHOR: Chen X L; Reindle A; Johnson E S (Reprint)
CORPORATE SOURCE: Thomas Jefferson Univ, Dept Biochem & Mol Pharmacol, 233 S 10th St, BLSB 231, Philadelphia, PA 19107 USA (Reprint); Thomas Jefferson Univ, Dept Biochem & Mol Pharmacol, Philadelphia, PA 19107 USA
COUNTRY OF AUTHOR: USA
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (MAY 2005) Vol. 25, No. 10, pp. 4311-4320.
Publisher: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA.
ISSN: 0270-7306.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 46
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI Misregulation of 2 mu m circle copy number in a SUMO pathway mutant

AB Attachment of the **ubiquitin**-like protein SUMO to other proteins is an essential process in Saccharomyces cerevisiae. However, yeast mutants lacking the SUMO ligases Siz1 and Siz2/Nfi1 are viable, even though they show dramatically reduced levels of SUMO conjugation. This siz1&UDelta; siz2&UDelta; double mutant is cold sensitive and has an unusual phenotype in that it forms irregularly shaped colonies that contain sectors of wild-type-appearing cells as well as sectors of enlarged cells that are arrested in G(2)/M. We have found that these phenotypes result from misregulation of the copy number of the endogenous yeast plasmid, the 2μ m circle. siz1&UDelta; siz2&UDelta; mutants have up to 40-fold-higher levels of 2μ m than do wild-type strains. Furthermore, 2μ m is responsible for the siz1&UDelta; siz2&UDelta; mutant's obvious growth defects, as siz1&UDelta; siz2&UDelta; [**cir** (0)] strains, which lack 2μ m, are no longer heterogeneous and show growth characteristics similar to those of the wild type. Possible mechanisms for SUMO's effect on 2μ m are suggested by the finding that both Flp1 recombinase and Rep2, two of the four proteins encoded by 2μ m, are covalently modified by SUMO. Our data suggest that SUMO attachment negatively regulates Flp1 levels, which may partially account for the

increased 2μ m copy number in the siz1&UDelta; siz2&UDelta; strain.

L11 ANSWER 5 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2005:304782 CAPLUS
TITLE: Jupiter's ammonia clouds-localized or **ubiquitous**?
AUTHOR(S): Atreya, S. K.; Wong, A. S.; Baines, K. H.; Wong, M. H.; Owen, T. C.
CORPORATE SOURCE: Department of Atmospheric, Oceanic, and Space Sciences, The University of Michigan, Ann Arbor, MI, 48109-2143, USA
SOURCE: Planetary and Space Science (2005), 53(5), 498-507
CODEN: PLSSAE; ISSN: 0032-0633
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Jupiter's ammonia clouds-localized or **ubiquitous**?
AB From an anal. of the Galileo Near IR Imaging Spectrometer (NIMS) data, Baines et al. (Icarus 159 (2002) 74) have reported that spectrally identifiable ammonia clouds (SIACs) cover less than 1% of Jupiter. Localized ammonia clouds have been identified also in the Cassini Composite IR Spectrometer (**CIRS**) observations (Planet. Space Sci. 52 (2004a) 385). Yet, ground-based, satellite and spacecraft observations show that clouds exist everywhere on Jupiter. Thermochem. models also predict that Jupiter must be covered with clouds, with the top layer made up of ammonia ice. For a solar composition atmospheric, models predict the base of the ammonia clouds to be at 720 mb, at 1000 mb if N/H were 4+solar, and at 0.5 bar for depleted ammonia of 10-2+solar (Planet. Space Sci. 47 (1999) 1243). Thus, the above NIMS and **CIRS** findings are seemingly at odds with other observations and cloud physics models. We suggest that the clouds of ammonia ice are **ubiquitous** on Jupiter, but that spectral identification of all but the freshest of the ammonia clouds and high altitude ammonia haze is inhibited by a combination of (i) dusting, starting with hydrocarbon haze particles falling from Jupiter's stratosphere and combining with an even much larger source-the hydrazine haze; (ii) cloud properties, including ammonia aerosol particle size effects. In this paper, we investigate the role of photochem. haze and find that a substantial amount of haze material can deposit on the upper cloud layer of Jupiter, possibly enough to mask its spectral signature. The stratospheric haze particles result from condensation of polycyclic aromatic hydrocarbons (PAHs), whereas hydrazine ice is formed from ammonia photochem. We anticipate similar conditions to prevail on Saturn.

L11 ANSWER 6 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2005:156681 CAPLUS
Correction of: 2005:60757
DOCUMENT NUMBER: 142:216629
Correction of: 142:132329
TITLE: Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:		
US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812777	A	20040330

TI Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular hyperlipidemia, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen.

L11 ANSWER 7 OF 81 CAPLUS DUPLICATE 5

ACCESSION NUMBER:	2005:139371 CAPLUS
DOCUMENT NUMBER:	142:195820
TITLE:	Gene expression profiles and biomarkers for the detection of Chagas disease and other disease-related gene transcripts in blood
INVENTOR(S):	Liew, Choong-Chin
PATENT ASSIGNEE(S):	Chondrogene Limited, Can.
SOURCE:	U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 802,875. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	42

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241729	A1	20041202	US 2004-813097	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241729	A1	20041202	US 2004-813097	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:		
US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2004-813097	A	20040330
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325

TI Gene expression profiles and biomarkers for the detection of Chagas disease and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular Chagas disease, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L11 ANSWER 8 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 6

ACCESSION NUMBER:	2005:112850 CAPLUS
DOCUMENT NUMBER:	142:153469
TITLE:	Gene expression profiles and biomarkers for the detection of lung disease-related and other disease-related gene transcripts in blood
INVENTOR(S):	Liew, Choong-chin
PATENT ASSIGNEE(S):	Chondrogene Limited, Can.
SOURCE:	U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875. CODEN: USXXCO
DOCUMENT TYPE:	Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241728	A1	20041202	US 2004-812764	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241728	A1	20041202	US 2004-812764	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2004-812764	A	20040330
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325

TI Gene expression profiles and biomarkers for the detection of lung disease-related and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L11 ANSWER 9 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:838610 CAPLUS
DOCUMENT NUMBER: 141:312238
TITLE: DNA microarray analysis of gene expression in the diagnosis of estrogen receptor positive- and negative-breast cancer
INVENTOR(S): Erlander, Mark G.; Ma, Xiao-Jun; Wang, Wei; Wittliff, James L.
PATENT ASSIGNEE(S): Arcturus Bioscience, Inc., USA
SOURCE: PCT Int. Appl., 226 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004079014	A2	20040916	WO 2002-XA2004006736	20040304
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, CN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2004079014	A2	20040916	WO 2004-US6736	20040304
WO 2004079014	A3	20050331		
W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX, MZ, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2003-451942P P 20030304
WO 2004-US6736 A 20040304

TI DNA microarray analysis of gene expression in the diagnosis of estrogen receptor positive- and negative-breast cancer

AB The invention relates to the identification and use of gene expression profiles, or patterns, suitable for identification of populations that are pos. and neg. for estrogen receptor expression. The gene expression profiles may be embodied in nucleic acid expression, protein expression, or other expression formats, and may be used in the study and/or diagnosis of cells and tissue in breast cancer as well as for the study and/or determination of prognosis of a patient, including breast cancer survival.

L11 ANSWER 10 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:681671 CAPLUS
DOCUMENT NUMBER: 141:201315
TITLE: Human host nucleic acid and protein sequences related to infectious disease and their use in decreasing viral infection and screening
INVENTOR(S): Hodge, Thomas W.; Morey, Natalie J.; Rubin, Don; Shaw, Michael W.; Sanchez, Anthony
PATENT ASSIGNEE(S): The Government of the United States of America as Represented by the Secretary of the Department of Health and Human Services, Centers for Disease Contol and Prevention, USA
SOURCE: PCT Int. Appl., 396 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004070002	A2	20040819	WO 2003-US37143	20031118

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2002-427464P P 20021118
US 2003-482604P P 20030625

TI Human host nucleic acid and protein sequences related to infectious disease and their use in decreasing viral infection and screening

AB Host nucleic acids and host proteins that participate in viral infection, such as human immunodeficiency virus (HIV-1 and HIV-2), influenza A, and Ebola virus, but whose inactivation is not lethal to the host cell, are identified by the gene trap method. A Moloney murine leukemia virus-derived shuttle vector that encodes for a promoterless neomycin-resistance gene integrates into the host genome at transcriptionally active genes, thereby disrupting the host gene but utilizing the host promoter to drive neomycin resistance carried by the vector. Cells surviving viral infection carry an interrupted host gene that is needed during the viral life cycle. Since the construct is a shuttle vector, it can function as a plasmid and can be moved from mammalian to bacterial systems, facilitating subcloning and DNA sequencing. Interfering with or disrupting the interaction between a host nucleic acid or host protein and a virus or viral protein confers an inhibition of or resistance to infection. Thus, interfering with such an interaction in a host subject can confer a therapeutic or prophylactic effect against a virus. The sequences identified can be used to identify agents that reduce or inhibit viral infection. Silent intefering double-stranded RNAs (siRNAs) are provide that recognize Rab9, AXL receptor tyrosine kinase, or other genes as a target sequence are provided. Expression of Rab9 siRNA decreases formation of lipid rafts (liquid-ordered microdomains enriched in sphingolipids and cholesterol), suggesting that viruses take advantage of rafts for completion of some steps of their replication cycle, and thus providing addnl. targets for antiviral action.

L11 ANSWER 11 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:633950 CAPLUS
DOCUMENT NUMBER: 141:169975
TITLE: Purification, cloning and characterization of L-amino acid oxidase with cytotoxic activity from Aplysia punctata and use for the diagnosis and treatment of cancer
INVENTOR(S): Butzke, Daniel; Goedert, Sigrid; Dittrich, Michael; Rudel, Thomas; Meyer, Thomas F.
PATENT ASSIGNEE(S): Max-Planck-Gesellschaft Zur Foerderung Der Wissenschaften E.V., Germany
SOURCE: PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004065415	A2	20040805	WO 2004-EP423	20040120
WO 2004065415	A3	20050120		

W: AE, AE, AG, AL, AL, AM, AM, AM, AT, AT, AU, AZ, AZ, BA, BB, BG, BG, BR, BR, BW, BY, BY, BZ, BZ, CA, CH, CN, CN, CO, CO, CR, CR, CU, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EC, EE, EE, EG, ES, ES, FI, FI, GB, GD, GE, GE, GH, GM, HR, HR, HU, HU, ID, IL, IN, IS, JP, JP, KE, KE, KG, KG, KP, KP, KP, KR, KR, KZ, KZ, KZ, LC, LK, LR, LS, LS, LT, LU, LV, MA, MD, MD, MG, MK, MN, MW, MX, MX, MZ, MZ, NA, NI

PRIORITY APPLN. INFO.: EP 2003-1232 A 20030120
EP 2003-26613 A 20031119

TI Purification, cloning and characterization of L-amino acid oxidase with cytotoxic activity from Aplysia punctata and use for the diagnosis and treatment of cancer

AB The present invention relates to a cytotoxic polypeptide which is an L-amino acid oxidase isolated from the ink of the sea hare Aplysia punctata via anion exchange chromatog. and gel filtration. The polypeptide is termed APIT (Aplysia punctata ink toxin). Tumor cells treated with APIT displays a morphol. which is neither typical for apoptosis nor for necrosis but rather is typical for oxidative damage induced cell death. The cDNA sequence and the encoded amino acid sequence of APIT isoforms are provided. The toxic and enzymic activity of APIT is due to the presence of an attached FAD. It was demonstrated that the cytotoxic activity depended on the H2O2 producing enzymic activity of APIT. From all amino acids tested only L-lysine and L-arginine served as substrates for APIT to produce hydrogen peroxide. Sensitivity of different tumor cell lines to APIT induced cell death was studied. Change in protein expression pattern in Jurkat T cells after treatment with APIT was investigated. The influence of APIT on the gene exression of tumor cells was investigated by Microarray technol. It was shown that healthy human cells are resistant against the APIT-induced cell death. APIT can be used for the manufacture of a medicament for the diagnosis and treatment of cancer.

L11 ANSWER 12 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:413134 CAPLUS
DOCUMENT NUMBER: 141:3254
TITLE: Substrates of N-end rule **ubiquitylation** and methods for measuring **ubiquitylation** of these substrates
INVENTOR(S): Davydov, Llia; Kenten, John H.; Biebuyck, Hans; Oberoi, Pankaj
PATENT ASSIGNEE(S): Meso Scale Technologies, LLC, USA
SOURCE: PCT Int. Appl., 137 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004042352	A2	20040521	WO 2003-US34148	20031028
WO 2004042352	A3	20050224		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004137597 A1 20040715 US 2003-693999 20031028

PRIORITY APPLN. INFO.: US 2002-422448P P 20021030

US 2003-486529P P 20030712

TI Substrates of N-end rule **ubiquitylation** and methods for measuring **ubiquitylation** of these substrates

AB The invention relates to methods, compns., compds. and kits for detecting, measuring and modulating protein **ubiquitylation** via the N-end rule pathway and for identifying substrates, enzymes and modulators of N-end rule **ubiquitylation**. The invention also claims specific substrates of N-end rule **ubiquitylation** as well as activated fragments of these substrates, proteases that expose N-degrons in these substrates, **ubiquitin** ligases that **ubiquitylate** these substrates and inhibitors of the **ubiquitylation** of these substrates. The invention claims complexes formed in vitro between **ubiquitin** and the following proteins: apraxin, tau, **SLP**, **HMG17**, **PinX1**, **CIR**, Cullin 3, **HMGN3**, **HSPC144**, and CDC6, where these complexes have higher specific activity than complexes from cell lysates, in cells, or in tissues. E3 **ubiquitin** ligases belong to a variety of different structural classes (e.g. HECT and RING finger) and act via a number of distinct pathways. Most E3 proteins that have been shown to interact with E2s and to mediate **ubiquitylation** in in vitro systems lack defined substrates other than themselves. One important **ubiquitylation** pathway is N-end rule **ubiquitylation** and some N-end rule substrates are linked to disease or pathol.

L11 ANSWER 13 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:449883 CAPLUS

DOCUMENT NUMBER: 140:402911

TITLE: Binary prediction tree modeling with many predictors and its uses in clinical and genomic applications

INVENTOR(S): Nevins, Joseph R.; West, Mike; Huang, Andrew T.

PATENT ASSIGNEE(S): Duke University, USA

SOURCE: PCT Int. Appl., 886 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 5

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004038376	A2	20040506	WO 2003-XA33946	20031024

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2004038376 A2 20040506 WO 2003-US33946 20031024

WO 2004038376 A3 20040826

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,

LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ,
OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM,
TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 2002-420729P	P	20021024
US 2002-421062P	P	20021025
US 2002-421102P	P	20021025
US 2002-424701P	P	20021108
US 2002-424715P	P	20021108
US 2002-424718P	P	20021108
US 2002-425256P	P	20021112
US 2003-448461P	P	20030221
US 2003-448462P	P	20030221
US 2003-457877P	P	20030327
US 2003-458373P	P	20030331
WO 2003-US33946	A	20031024

TI Binary prediction tree modeling with many predictors and its uses in clinical and genomic applications

AB The statistical anal. described and claimed is a predictive statistical tree model that overcomes several problems observed in prior statistical models and regression analyses, while ensuring greater accuracy and predictive capabilities. Although the claimed use of the predictive statistical tree model described herein is directed to the prediction of a disease in individuals, the claimed model can be used for a variety of applications including the prediction of disease states, susceptibility of disease states or any other biol. state of interest, as well as other applicable non-biol. states of interest. This model first screens genes to reduce noise, applies kmeans correlation-based clustering targeting a large number of clusters, and then uses singular value decompns. (SVD) to extract the single dominant factor (principal component) from each cluster. This generates a statistically significant number of cluster-derived singular factors, that are referred to as metagenes, that characterize multiple patterns of expression of the genes across samples. The strategy aims to extract multiple such patterns while reducing dimension and smoothing out gene-specific noise through the aggregation within clusters. Formal predictive anal. then uses these metagenes in a Bayesian classification tree anal. This generates multiple recursive partitions of the sample into subgroups (the 'leaves' of the classification tree), and assocs. Bayesian predictive probabilities of outcomes with each subgroup. Overall predictions for an individual sample are then generated by averaging predictions, with appropriate wts., across many such tree models. The model includes the use of iterative out-of-sample, cross-validation predictions leaving each sample out of the data set one at a time, refitting the model from the remaining samples and using it to predict the hold-out case. This rigorously tests the predictive value of a model and mirrors the real-world prognostic context where prediction of new cases as they arise is the major goal.

L11 ANSWER 14 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	2005:1997 CAPLUS
DOCUMENT NUMBER:	142:111841
TITLE:	Gene expression profiles and biomarkers for the detection of depression-related and other disease-related gene transcripts in blood
INVENTOR(S):	Liew, Choong-Chin
PATENT ASSIGNEE(S):	Chondrogene Limited, Can.
SOURCE:	U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 802,875. CODEN: USXXCO
DOCUMENT TYPE:	Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004265868	A1	20041230	US 2004-812702	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
US 2004265868	A1	20041230	US 2004-812702	20040330
US 2004265868	A1	20041230	US 2004-812702	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812702	A	20040330

TI Gene expression profiles and biomarkers for the detection of depression-related and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular mental depression, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen.

L11 ANSWER 15 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:60754 CAPLUS
Correction of: 2004:1036571
DOCUMENT NUMBER: 142:233342
Correction of: 142:16836
TITLE: Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 156 pp., Cont.-in-part of U.S.

Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004241727	A1	20041202	US 2004-812731	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812731	A	20040330

TI Sequences of human schizophrenia related genes and use for diagnosis, prognosis and therapy

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L11 ANSWER 16 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:60755 CAPLUS
Correction of: 2004:1036570
DOCUMENT NUMBER: 142:154259
Correction of: 142:36938
TITLE: Analysis of genetic information contained in peripheral blood for diagnosis, prognosis and monitoring treatment of allergy, infection and genetic disease in human
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.

SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812707	A	20040330

TI Analysis of genetic information contained in peripheral blood for diagnosis, prognosis and monitoring treatment of allergy, infection and genetic disease in human

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular allergy, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in hypertension, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular treatment regimen. [This abstract record is one of 3 recordsfor this document necessitated by thelarge number of index entries required tofully index the document and publicationsystem constraints.].

L11 ANSWER 17 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:85984 CAPLUS
DOCUMENT NUMBER: 140:194432

TITLE: Human prostate cancer marker genes associated with various metastatic stages identified by gene profiling, and related compositions, kits, and methods for diagnosis, prognosis and therapy
INVENTOR(S): Schlegel, Robert; Endege, Wilson O.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 131 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2004009481	A1	20040115	US 2002-166883		20020611
US 2004009481	A1	20040115	US 2002-166883		20020611
PRIORITY APPLN. INFO.:			US 2001-297285P	P	20010611
			US 2002-166883	A	20020611

TI Human prostate cancer marker genes associated with various metastatic stages identified by gene profiling, and related compositions, kits, and methods for diagnosis, prognosis and therapy

AB The invention relates to compns., kits, and methods for diagnosing, staging, prognosing, monitoring and treating human prostate cancers. A variety of marker genes are provided, wherein changes in the levels of expression of one or more of the marker genes is correlated with the presence of prostate cancer. In particular, three sets of the marker genes set, corresponding to 11617 GenBank Accession Nos. (only 2168 new submissions) and 15 SEQ IDs, are identified by transcription profiling using RNA derived from clin. samples, that were expressed at least 2-fold or greater than the normal controls. Using TNM staging approach, these markers are divided to three groups, ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the liver (M stage); ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the bone (M stage); and ones can be used to determine whether prostate cancer has metastasized, or is likely to metastasize, to the lymph nodes (N stage and/or M stage). The invention also relates to a kit for assessing the specific type of metastatic prostate cancer, e.g., cancer that has metastasized to the liver, bone or lymph nodes. [This abstract record is one of three records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L11 ANSWER 18 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2004:481334 CAPLUS
DOCUMENT NUMBER: 141:169678
TITLE: Identification and Characterization of a Novel BASH N Terminus-associated Protein, BNAS2
AUTHOR(S): Imamura, Yasuhiro; Katahira, Takashi; Kitamura, Daisuke
CORPORATE SOURCE: Research Institute for Biological Sciences, Tokyo University of Science, Chiba, 278-0022, Japan
SOURCE: Journal of Biological Chemistry (2004), 279(25), 26425-26432
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Identification and Characterization of a Novel BASH N Terminus-associated Protein, BNAS2

AB A B cell-specific adaptor protein, BASH (also known as BLNK or **SLP** -65), is crucial for B cell receptor (BCR) signaling. BASH binds to

various signaling intermediates, such as Btk, PLCγ2, Vav, and Grb2, through its well defined motifs. Although functional significance of such interactions has been documented, BASH-mediated signal transduction mechanism is not fully understood. Using the yeast two-hybrid system, we have identified a novel protein that binds to a conserved N-terminal domain of BASH, which we named BNAS2 (BASH N terminus associated protein 2). From its deduced amino acid sequence, BNAS2 is presumed to contain four transmembrane domains, which are included in a central MARVEL domain, and to localize to endoplasmic reticulum. BNAS2 was co-precipitated with BASH as well as Btk and ERK2 from a lysate of mouse B cell line. In the transfected cells, the exogenous BNAS2 was localized in a mesh-like structure in the cytoplasm resembling that of endoplasmic reticulum (ER) and nuclear membrane. BASH was co-localized with BNAS2 in a manner dependent on its N-terminal domain. RT-PCR anal. indicated that BNAS2 mRNA is expressed **ubiquitously** except for plasma cells. In chicken B cell line DT40, overexpression of BNAS2 resulted in an enhancement of BCR ligation-mediated transcriptional activation of Elk1, but not of NF-κB, in a manner dependent on the dose of BNAS2. Thus BNAS2 may serve as a scaffold for signaling proteins such as BASH, Btk, and ERK at the ER and nuclear membrane and may facilitate ERK activation by signaling from cell-surface receptors.

REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 19 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:746376 CAPLUS

DOCUMENT NUMBER: 141:325914

TITLE: Evidence for the notch signaling pathway on the role of estrogen in angiogenesis

AUTHOR(S): Soares, R.; Balogh, G.; Guo, S.; Gaertner, F.; Russo, J.; Schmitt, F.

CORPORATE SOURCE: Institute of Molecular Pathology and Immunology of the University of Porto (IPATIMUP), Medical Faculty, University of Porto, Oporto, 4200, Port.

SOURCE: Molecular Endocrinology (2004), 18(9), 2333-2343
CODEN: MOENEN; ISSN: 0888-8809

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Evidence for the notch signaling pathway on the role of estrogen in angiogenesis

AB We have investigated the mol. mechanisms involved in 17β-estradiol-induced angiogenic pathway. We show here that 17β-estradiol promoted a 6-fold increase in Jagged1 expression and an 8-fold increase in Notch1 expression by cDNA arrays in breast cancer MCF7 cells. Interestingly, Jagged1 was abrogated by incubation with the estrogen antagonist, ICI182,780. A similar upregulation of both Notch1 receptor and Jagged1 ligand was found in endothelial cells. Addnl., imperfect estrogen-responsive elements were found in the 5' untranslated region of Notch1 and Jagged1 genes. Treatment with 17β-estradiol also led to an activation of Notch signaling in MCF7 cells expressing Notch1 reporter gene or by promoting Jagged1-induced Notch signaling in coculture assays. Inoculation of MCF7 cells in 17β-estradiol-treated nude mice resulted in up-regulation of Notch1 expression as well as increased number of tumor microvessels in comparison to placebo-treated mice. Notch1-expressing endothelial cell cultures formed cord-like structures on Matrigel in contrast to cells expressing a dominant-neg. form of Notch1, emphasizing the relevance of Notch1 pathway in vessel assembly. Finally, Notch1-expressing MCF7 cells up-regulated hypoxia-inducible factor 1α gene, a well-known angiogenic factor that clustered with Notch1 gene. This study implicates Notch signaling in the cross talk between 17β-estradiol and angiogenesis.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 20 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 2004:1056406 CAPLUS
DOCUMENT NUMBER: 142:73079
TITLE: Grb2 and the non-T cell activation linker NTAL constitute a Ca2+-regulating signal circuit in B lymphocytes
AUTHOR(S): Stork, Bjoern; Engelke, Michael; Frey, Juergen; Horejsi, Vaclav; Hamm-Baarke, Andrea; Schraven, Burkhart; Kurosaki, Tomohiro; Wienands, Juergen
CORPORATE SOURCE: Department of Biochemistry and Molecular Immunology, University of Bielefeld, Bielefeld, 33615, Germany
SOURCE: Immunity (2004), 21(5), 681-691
CODEN: IUNIEH; ISSN: 1074-7613
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Grb2 and the non-T cell activation linker NTAL constitute a Ca2+-regulating signal circuit in B lymphocytes
AB Activation of the B cell antigen receptor triggers phosphorylation of cytoplasmic and transmembrane adaptor proteins such as **SLP**-65 and NTAL, resp. Specific phosphoacceptor sites in **SLP**-65 serve as docking sites for Ca2+-mobilizing enzymes Btk and PLC-γ2. Phosphorylated NTAL recruits the Grb2 linker, but downstream signaling cascades are unclear. The authors now show that receptor-induced tyrosine phosphorylation of NTAL and concomitant Grb2 complex formation critically modulate the Ca2+ response without affecting **SLP**-65 and PLC-γ2 phosphorylation. Grb2 turned out to play a neg. regulatory role, which appears to be eliminated upon binding to NTAL. This allows for a sustained release of intracellular Ca2+ and is mandatory for subsequent entry of Ca2+ from extracellular sources. Thus, elevation of Ca2+ is regulated by at least two signaling modules, the B cell-specific Ca2+ initiation complex comprising **SLP**-65, Btk, and PLC-γ2 and the more **ubiquitously** expressed NTAL/Grb2 complex, which acts as an amplifier by switching off inhibitory elements.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 21 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:102214 CAPLUS
DOCUMENT NUMBER: 140:126882
TITLE: Regulation of ZAP-70 activation and TCR signaling by two related proteins, Sts-1 and Sts-2
AUTHOR(S): Carpino, Nick; Turner, Steve; Mekala, Divya; Takahashi, Yutaka; Zang, Heesuk; Geiger, Terrence L.; Doherty, Peter; Ihle, James N.
CORPORATE SOURCE: Department of Biochemistry, Saint Jude Children's Research Hospital, Memphis, TN, 38105, USA
SOURCE: Immunity (2004), 20(1), 37-46
CODEN: IUNIEH; ISSN: 1074-7613
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Regulation of ZAP-70 activation and TCR signaling by two related proteins, Sts-1 and Sts-2
AB T cells play a central role in the recognition and elimination of foreign pathogens. Signals through the T cell receptor (TCR) control the extent and duration of the T cell response. To ensure that T cells are not inappropriately activated, signaling pathways downstream of the TCR are subject to multiple levels of pos. and neg. regulation. Herein, the authors describe two related proteins, Sts-1 and Sts-2, that neg. regulate TCR signaling. T cells from mice lacking Sts-1 and Sts-2 are

hyperresponsive to TCR stimulation. The phenotype is accompanied by increased Zap-70 phosphorylation and activation, including its **ubiquitinated** forms. Addnl., hyperactivation of signaling proteins downstream of the TCR, a marked increase in cytokine production by Sts1/2-/- T cells, and increased susceptibility to autoimmunity in a mouse model of multiple sclerosis is observed Therefore, Sts-1 and Sts-2 are critical regulators of the signaling pathways that regulate T cell activation.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 22 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:906937 CAPLUS

DOCUMENT NUMBER: 142:2463

TITLE: GSK3-mediated BCL-3 phosphorylation modulates its degradation and its oncogenicity

AUTHOR(S): Viatour, Patrick; Dejardin, Emmanuel; Warnier, Michael; Lair, Florence; Claudio, Estefania; Bureau, Fabrice; Marine, Jean-Christophe; Merville, Marie-Paule; Maurer, Ulrich; Green, Douglas; Piette, Jacques; Siebenlist, Ulrich; Bours, Vincent; Chariot, Alain

CORPORATE SOURCE: Laboratory of Medical Chemistry and Human Genetics, Center for Biomedical Integrated Genoproteomics, University of Liege, Liege, 4000, Belg.

SOURCE: Molecular Cell (2004), 16(1), 35-45
CODEN: MOCEFL; ISSN: 1097-2765

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English

TI GSK3-mediated BCL-3 phosphorylation modulates its degradation and its oncogenicity

AB The oncoprotein BCL-3 is a nuclear transcription factor that activates NF-κB target genes through formation of heterocomplexes with p50 or p52. BCL-3 is phosphorylated in vivo, but specific BCL-3 kinases have not been identified so far. The authors show that BCL-3 is a substrate for the protein kinase GSK3 and that GSK3-mediated BCL-3 phosphorylation, which is inhibited by Akt activation, targets its degradation through the proteasome pathway. This phosphorylation modulates its association with HDAC1, -3, and -6 and attenuates its oncogenicity by selectively controlling the expression of a subset of newly identified target genes such as SLPI and Cxcl1. The authors' results therefore suggest that constitutive BCL-3 phosphorylation by GSK3 regulates BCL-3 turnover and transcriptional activity.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 23 OF 81 MEDLINE on STN

ACCESSION NUMBER: 2004336526 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15238603

TITLE: Inactivation of c-Cbl reverses neonatal lethality and T cell developmental arrest of **SLP**-76-deficient mice.

AUTHOR: Chiang Y Jeffrey; Sommers Connie L; Jordan Martha S; Gu Hua; Samelson Lawrence E; Koretzky Gary A; Hodes Richard J

CORPORATE SOURCE: Experimental Immunology Branch, Building 10, Room 4B36, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA.. chiangj@mail.nih.gov

SOURCE: Journal of experimental medicine, (2004 Jul 5) 200 (1) 25-34.
Journal code: 2985109R. ISSN: 0022-1007.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200410
ENTRY DATE: Entered STN: 20040708
Last Updated on STN: 20041027
Entered Medline: 20041026

TI Inactivation of c-Cbl reverses neonatal lethality and T cell developmental arrest of **SLP**-76-deficient mice.

AB c-Cbl is an adaptor protein that negatively regulates signal transduction events involved in thymic-positive selection. To further characterize the function of c-Cbl in T cell development, we analyzed the effect of c-Cbl inactivation in mice deficient in the scaffolding molecule **SLP**-76. **SLP**-76-deficient mice show a high frequency of neonatal lethality; and in surviving mice, T cell development is blocked at the DN3 stage. Inactivation of c-cbl completely reversed the neonatal lethality seen in **SLP**-76-deficient mice and partially reversed the T cell development arrest in these mice. **SLP**-76(-/-) Cbl(-/-) mice exhibited marked expansion of polarized T helper type (Th)1 and Th2 cell peripheral CD4(+) T cells, lymphoid infiltrates of parenchymal organs, and premature death. This rescue of T cell development is T cell receptor dependent because it does not occur in recombination activating gene 2(-/-) **SLP**-76(-/-) Cbl(-/-) triple knockout mice. Analysis of the signal transduction properties of **SLP**-76(-/-) Cbl(-/-) T cells reveals a novel **SLP**-76- and linker for activation of T cells-independent pathway of extracellular signal-regulated kinase activation, which is normally down-regulated by c-Cbl.

L11 ANSWER 24 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:942764 CAPLUS
DOCUMENT NUMBER: 140:3792
TITLE: Genes expressed in atherosclerotic tissue and their use in diagnosis and pharmacogenetics
INVENTOR(S): Nevins, Joseph; West, Mike; Goldschmidt, Pascal
PATENT ASSIGNEE(S): Duke University, USA
SOURCE: PCT Int. Appl., 408 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003091391	A2	20031106	WO 2002-XA38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003091391	A2	20031106	WO 2002-US38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF,				

CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:	US 2002-374547P	P	20020423
	US 2002-420784P	P	20021024
	US 2002-421043P	P	20021025
	US 2002-424680P	P	20021108
	WO 2002-US38221	A	20021112

TI Genes expressed in atherosclerotic tissue and their use in diagnosis and pharmacogenetics

AB Genes whose expression is correlated with an determinant of an atherosclerotic phenotype are provided. Also provided are methods of using the subject atherosclerotic determinant genes in diagnosis and treatment methods, as well as drug screening methods. In addition, reagents and kits thereof that find use in practicing the subject methods are provided. Also provided are methods of determining whether a gene is correlated with a disease phenotype, where correlation is determined using a Bayesian anal.

L11 ANSWER 25 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	2003:409169 CAPLUS
DOCUMENT NUMBER:	138:380506
TITLE:	Genes that are differentially expressed during erythropoiesis and their diagnostic and therapeutic uses
INVENTOR(S):	Brissette, William H.; Neote, Kuldeep S.; Zagouras, Panayiotis; Zenke, Martin; Lemke, Britt; Hacker, Christine
PATENT ASSIGNEE(S):	Pfizer Products Inc., USA; Max-Delbrueck-Centrum Fuer Molekulare Medizin
SOURCE:	PCT Int. Appl., 285 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	2
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003038130	A2	20030508	WO 2002-XA34888	20021031
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003038130	A2	20030508	WO 2002-US34888	20021031
WO 2003038130	A3	20040212		
WO 2003038130	C1	20040422		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2001-335048P P 20011031
US 2001-335183P P 20011102
WO 2002-US34888 A 20021031

TI Genes that are differentially expressed during erythropoiesis and their diagnostic and therapeutic uses

AB The present invention provides mol. targets that regulate erythropoiesis. Groups of genes or their encoded gene products comprise panels of the invention and may be used in therapeutic intervention, therapeutic agent screening, and in diagnostic methods for diseases and/or disorders of erythropoiesis. The panels were discovered using gene expression profiling of erythroid progenitors with Affymetrix HU6800 and HG-U95Av2 chips. Cells from an in vitro growth and differentiation system of SCF-Epo dependent human erythroid progenitors, E-cadherin+/CD36+ progenitors, cord blood, or CD34+ peripheral blood stem cells were analyzed. The HU6800 chip contains probes from 13,000 genes with a potential role in cell growth, proliferation, and differentiation and the HG-U95Av2 chip contains 12,000 full-length, functionally-characterized genes. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L11 ANSWER 26 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:662654 CAPLUS
DOCUMENT NUMBER: 139:301507
TITLE: Identification and Characterization of a Cell Cycle and Apoptosis Regulatory Protein-1 as a Novel Mediator of Apoptosis Signaling by Retinoid CD437
AUTHOR(S): Rishi, Arun K.; Zhang, Liyue; Boyanapalli, Madanamohan; Wali, Anil; Mohammad, Ramzi M.; Yu, Yingjie; Fontana, Joseph A.; Hatfield, James S.; Dawson, Marcia I.; Majumdar, Adhip P. N.; Reichert, Uwe
CORPORATE SOURCE: Department of Internal Medicine and Karmanos Cancer Institute, Veterans Affairs Medical Center, Wayne State University, Detroit, MI, 48201, USA
SOURCE: Journal of Biological Chemistry (2003), 278(35), 33422-33435
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Identification and Characterization of a Cell Cycle and Apoptosis Regulatory Protein-1 as a Novel Mediator of Apoptosis Signaling by Retinoid CD437

AB CD437, a novel retinoid, causes cell cycle arrest and apoptosis in a number of cancer cells including human breast carcinoma (HBC) by utilizing an undefined retinoic acid receptor/retinoid X receptor-independent mechanism. To delineate mediators of CD437 signaling, we utilized a random antisense-dependent functional knockout genetic approach. We identified a cDNA that encodes .apprx.130-kDa HBC cell perinuclear protein (termed CARP-1). Treatments with CD437 or chemotherapeutic agent adriamycin, as well as serum deprivation of HBC cells, stimulate CARP-1 expression. Reduced levels of CARP-1 result in inhibition of apoptosis by CD437 or adriamycin, whereas increased expression of CARP-1 causes elevated levels of cyclin-dependent kinase inhibitor p21WAF1/CIP1 and apoptosis. CARP-1 interacts with 14-3-3 protein as well as causes reduced expression of cell cycle regulatory genes including c-Myc and cyclin B1. Loss of c-Myc sensitizes cells to apoptosis by CARP-1, whereas expression of c-Myc or 14-3-3 inhibits CARP-1-dependent apoptosis. Thus, apoptosis induction by CARP-1 involves sequestration of 14-3-3 and CARP-1-mediated altered expression of multiple cell cycle regulatory genes. Identification of CARP-1 as a key mediator of signaling by CD437 or

adriamycin allows for delineation of pathways that, in turn, may prove beneficial for design and targeting of novel antitumor agents.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 27 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:193558 SCISEARCH
THE GENUINE ARTICLE: 774ZN
TITLE: Transition to solar minimum at high solar latitudes: Energetic particles from corotating interaction regions
AUTHOR: Hofer M Y (Reprint); Marsden R G; Sanderson T R; Tranquille C; Forsyth R J
CORPORATE SOURCE: Estec, ESA, Res & Sci Support Dept, Keplerlaan 1, NL-2201 AZ Noordwijk, Netherlands (Reprint); Estec, ESA, Res & Sci Support Dept, NL-2201 AZ Noordwijk, Netherlands; Univ London Imperial Coll Sci Technol & Med, Blackett Lab, London, England
COUNTRY OF AUTHOR: Netherlands; England
SOURCE: GEOPHYSICAL RESEARCH LETTERS, (11 SEP 2003) Vol. 30, No. 19, arn. 8034.
Publisher: AMER GEOPHYSICAL UNION, 2000 FLORIDA AVE NW, WASHINGTON, DC 20009 USA.
ISSN: 0094-8276.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 15
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
TI Transition to solar minimum at high solar latitudes: Energetic particles from corotating interaction regions
AB One of the key questions to be addressed during the solar maximum phase of the Ulysses mission has been the nature of the **ubiquitous** energetic particle populations observed at all helio-latitudes from equator to poles. During the current, post-maximum phase of its mission, Ulysses has encountered the return to more stable solar wind, high speed stream structure, leading to the formation of stream and/or corotating interaction regions, in addition to the coronal mass ejection associated transients. The analysis reported here presents the identification of the first **CIR** after the recent solar maximum recorded by Ulysses COSPIN/LET at high helio-latitudes based on energetic particle composition data recorded in the similar to5-30 MeV/n range. The recurrent compression region appears during 3 6 solar rotations in 2002 and is observed from approximate to52degreesN to approximate to37degreesN heliographic latitude at heliocentric distances from approximate to3.1 AU to approximate to3.8 AU.

L11 ANSWER 28 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:996915 SCISEARCH
THE GENUINE ARTICLE: 741JN
TITLE: **Ubiquitous** computing in home networks
AUTHOR: Schulzrinne H (Reprint); Wu X T; Sidiroglou S; Berger S
CORPORATE SOURCE: Columbia Univ, Dept Elect Engn, New York, NY 10027 USA (Reprint); Columbia Univ, Dept Comp Sci, New York, NY 10027 USA
COUNTRY OF AUTHOR: USA
SOURCE: IEEE COMMUNICATIONS MAGAZINE, (NOV 2003) Vol. 41, No. 11, pp. 128-135.
Publisher: IEEE-INST ELECTRICAL ELECTRONICS ENGINEERS INC, 445 HOES LANE, PISCATAWAY, NJ 08855 USA.
ISSN: 0163-6804.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English

REFERENCE COUNT: 18

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI **Ubiquitous** computing in home networks

AB In the past decade, there have been numerous efforts in **ubiquitous** computing. For home networks, we believe that **ubiquitous** computing requires a global-scale system that is securable, administered by multiple independent nonspecialist administrators, and integrates off-the-shelf hardware and software. In this system every home owner acts as an administrator of the network in the home. We are developing such a system based on Session Initiation Protocol (SIP), with Bluetooth devices for location sensing and Service Location Protocol (**SLP**) for service discovery. We also introduce context-aware location information to augment device discovery and user communication. The system builds on our CINEMA infrastructure.

L11 ANSWER 29 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:504470 SCISEARCH

THE GENUINE ARTICLE: 688HD

TITLE: S1P(3) receptors mediate the potent constriction of cerebral arteries by sphingosine-1-phosphate

AUTHOR: Salomone S; Yoshimura S; Reuter U; Foley M; Thomas S S; Moskowitz M A; Waeber C (Reprint)

CORPORATE SOURCE: Harvard Univ, Massachusetts Gen Hosp, Sch Med, Dept Radiol, Stroke & Neurovasc Regulat Lab, CNY 149 13th St, Room 6403, Charlestown, MA 02129 USA (Reprint); Harvard Univ, Massachusetts Gen Hosp, Sch Med, Dept Radiol, Stroke & Neurovasc Regulat Lab, Charlestown, MA 02129 USA; Catania Univ, Sch Med, Dept Pharmacol, Catania, Italy

COUNTRY OF AUTHOR: USA; Italy

SOURCE: EUROPEAN JOURNAL OF PHARMACOLOGY, (23 MAY 2003) Vol. 469, No. 1-3, pp. 125-134.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0014-2999.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI S1P(3) receptors mediate the potent constriction of cerebral arteries by sphingosine-1-phosphate

AB We characterized the effect of Sphingosine-1-phosphate (SIP) on vascular tone. SIP selectively constricted isolated cerebral, but not peripheral arteries, despite **ubiquitous** expression of S1P(1), S1P(2), S1P(3) and S1P(5) receptor mRNA. Clostridium B and C3 toxins and the rhokinase inhibitor Y27632 (trans-N-(4-pyridyl)-4-(1-aminoethyl)-cyclohexane carboxamide) reduced this vasoconstriction to S1P, indicating that the response was mediated through Rho. Pertussis toxin displayed only weak inhibition, suggesting minor involvement of G(i/o) protein. The S1P effect was specifically reduced by adenovirus bearing a **slp**(3) but not **slp**(2), antisense construct. Furthermore, suramin, which selectively blocks S1P(3) receptors, inhibited the vasoconstrictor effect of S1P, indicating that S1P(3) receptors account for at least part of S1P-mediated vasoconstriction in cerebral arteries. In vivo, intracarotid injection of S1P decreased cerebral blood flow, an effect prevented by suramin treatment. Because SIP constricts cerebral blood vessels and is released from platelets during clotting, the S1P/S1P3 System constitutes a novel potential target for cerebrovascular disease therapy. (C) 2003 Elsevier Science B.V. All rights reserved.

L11 ANSWER 30 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:997356 CAPLUS

DOCUMENT NUMBER: 141:61060

TITLE: Transition to solar minimum at high solar latitudes: energetic particles from corotating interaction regions
AUTHOR(S): Hofer, M. Y.; Marsden, R. G.; Sanderson, T. R.; Tranquille, C.; Forsyth, R. J.
CORPORATE SOURCE: Research and Scientific Support Dept. of ESA, ESTEC, Noordwijk, Neth.
SOURCE: Geophysical Research Letters (2003), 30(19), ULY8/1-ULY8/4
CODEN: GPRLAJ; ISSN: 0094-8276
PUBLISHER: American Geophysical Union
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Transition to solar minimum at high solar latitudes: energetic particles from corotating interaction regions.

AB One of the key questions to be addressed during the solar maximum phase of the Ulysses mission was the nature of the **ubiquitous** energetic particle populations observed at all helio-latitudes from equator to poles. During the current, post-maximum phase of its mission, Ulysses has encountered the return to more stable solar wind, high speed stream structure, giving stream and/or corotating interaction regions, in addition to the coronal mass ejection associated transients. The anal. reported here presents the identification of the 1st **CIR** after the recent solar maximum recorded by Ulysses COSPIN/LET at high helio-latitudes based on energetic particle composition data recorded in the .apprx.5-30 MeV/n range. The recurrent compression region appears during 36 solar rotations in 2002 and is observed from ≈52°N to ≈37°N heliog. latitude at heliocentric distances from ≈3.1 AU to ≈3.8 AU.

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 31 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:832576 CAPLUS
DOCUMENT NUMBER: 137:346197
TITLE: Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 764 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085309	A2	20021031	WO 2002-US13143	20020423
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004049022	A1	20040311	US 2003-627930	20030725
PRIORITY APPLN. INFO.:			US 2001-286036P P	20010424

WO 2002-US13135 A2 20020423
WO 2002-US13143 A2 20020423

OTHER SOURCE(S): MARPAT 137:346197

TI Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent

AB This patent relates to a composition comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addition, they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the treatment of bronchoconstriction and/or inflammation. These agents and the composition and formulations provided are suitable for the treatment of respiratory tract, pulmonary and malignant diseases associated with bronchoconstriction, respiratory tract inflammation and allergies, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as allergies, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis, RDA, COPD, and cancers, among others. The present agents and composition may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L11 ANSWER 32 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:832575 CAPLUS
DOCUMENT NUMBER: 137:346196
TITLE: Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent
INVENTOR(S): Nyce, Jonathan W.; Li, Yukui; Sandrasagra, Anthony; Katz, Evan; Pabalan, Jonathan; Aguilar, Douglas; Miller, Shoreh; Tang, Lei; Shahabuddin, Syed
PATENT ASSIGNEE(S): Epigenesis Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 872 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002085308	A2	20021031	WO 2002-US13135	20020423
WO 2002085308	A3	20021219		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002085308 A2 20021031 WO 2002-XA13135 20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002085308 A2 20021031 WO 2002-XB13135 20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2002085308 A2 20021031 WO 2002-XC13135 20020423

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004049022 A1 20040311 US 2003-627930 20030725

PRIORITY APPLN. INFO.: US 2001-286137P P 20010424
WO 2002-US13135 A 20020423
WO 2002-US13143 A2 20020423

OTHER SOURCE(S): MARPAT 137:346196

TI Treatment of respiratory and lung diseases with antisense oligonucleotides and a bronchodilating agent

AB This patent relates to a composition comprising a carrier, oligonucleotides (oligos) that are antisense to adenosine receptors, and contain low amts. of or no adenosine (A), plus bronchodilating agents. All antisense oligonucleotides designed in accordance with the invention were highly effective at countering or reducing effects mediated by the receptors to which they are targeted. Two antisense phosphorothioated oligos targeting human adenosine A1 receptor mRNA, one targeting adenosine A2b receptor, and two targeting an A3 receptor are capable of countering the effect of exogenously administered adenosine which is mediated by the specific receptor they are targeted to. The activity of the antisense oligos are specific to the target and substitutively fail to inhibit another target. An oligonucleotide wherein the phosphodiester bonds are substituted with phosphorothioate bonds evidenced an unexpected superiority over the phosphodiester antisense oligo. In addition, they result in extremely low or non-existent deleterious side effects or toxicity. This represents 100% success in providing agents that are highly effective and specific in the treatment of bronchoconstriction and/or inflammation. Treatment with antisense oligonucleotides in combination with anti-inflammatory steroid and/or ubiquinones is also provided. These agents and the composition and formulations provided are suitable for the treatment of respiratory tract,

pulmonary and malignant diseases associated with bronchoconstriction, respiratory tract inflammation and allergies, impaired airways, including lung disease and diseases whose secondary effects afflict the lungs of a subject, such as allergies, asthma, impeded respiration, allergic rhinitis, pain, cystic fibrosis, pulmonary fibrosis, RDA, COPD, and cancers, among others. The present agents and composition may be administered preventatively, prophylactically or therapeutically in conjunction with other therapies, or may be utilized as a substitute for therapies that have significant, neg. side effects. The method of the present invention is also practiced with antisense oligonucleotides targeted to many genes, mRNAs and their corresponding proteins in essential the same manner.

L11 ANSWER 33 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:696159 CAPLUS
DOCUMENT NUMBER: 137:246071
TITLE: Gene expression profiles relating to normal and osteoarthritic cartilage
INVENTOR(S): Liew, Choong-Chin; Marshall, Wayne E.; Zhang, Hongwei
PATENT ASSIGNEE(S): Chondrogene Inc., Can.
SOURCE: PCT Int. Appl., 777 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 42
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070737	A2	20020912	WO 2002-CA247	20020228
WO 2002070737	C1	20021031		
WO 2002070737	C2	20031002		
WO 2002070737	A3	20040129		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2439504	AA	20020912	CA 2002-2439504	20020228
EP 1404868	A2	20040407	EP 2002-703416	20020228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2002007657	A	20041026	BR 2002-7657	20020228
JP 2004536575	T2	20041209	JP 2002-570759	20020228
US 2004248169	A1	20041209	US 2004-812737	20040330

PRIORITY APPLN. INFO.:

US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-85783	A2	20020228
WO 2002-CA247	W	20020228
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A3	20040312

TI Gene expression profiles relating to normal and osteoarthritic cartilage
AB The invention provides gene expression profiles comprising one or more polynucleotide sequences that are expressed in chondrocytes from any of the following developmental and disease stages: fetus, normal adult, mild

osteoarthritis, moderate osteoarthritis, marked osteoarthritis, and severe osteoarthritis. Complementary DNA libraries were constructed from human fetal, normal, mild osteoarthritic and severe osteoarthritic cartilage samples (13,398, 17,151, 12,651, and 14,222 expressed sequence tags (ESTs), resp.). The known and novel clones derived from these libraries were then used to construct human chondrocyte-specific microarrays to generate differential gene expression profiles useful as a diagnostic tools for detection of osteoarthritis. A total of 5807 expressed gene sequences are provided and matched to known gene sequences, other ESTs, or mitochondrial, ribosomal, vector, and cDNA/hypothetical protein sequences in the public databases. Arrays of the invention are useful as a gold standard for osteoarthritis diagnosis and for use to identify and monitor therapeutic efficacy of new drug targets.

L11 ANSWER 34 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:971332 SCISEARCH
THE GENUINE ARTICLE: 619GP
TITLE: c-Cbl and Cbl-b regulate T cell responsiveness by promoting ligand-induced TCR down-modulation
AUTHOR: Naramura M; Jang I K; Kole H; Huang F; Haines D; Gu H (Reprint)
CORPORATE SOURCE: NIAID, Immunol Lab, NIH, Rockville, MD 20852 USA (Reprint); NCI, Pathol Histotechnol Lab, Frederick Canc Res & Dev Ctr, Frederick, MD 21702 USA
COUNTRY OF AUTHOR: USA
SOURCE: NATURE IMMUNOLOGY, (DEC 2002) Vol. 3, No. 12, pp. 1192-1199.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707 USA.
ISSN: 1529-2908.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI c-Cbl and Cbl-b regulate T cell responsiveness by promoting ligand-induced TCR down-modulation

AB How Cbl family proteins regulate T cell responses is unclear. We found that c-Cbl Cbl-b double knockout (dKO) T cells became hyperresponsive upon anti-CD3 stimulation, even though the major T cell antigen receptor (TCR) signaling pathways were not enhanced. The dKO T cells did not down-modulate surface TCR after ligand engagement, which resulted in sustained TCR signaling. However, these cells showed normal ligand-independent TCR internalization, and trafficking of internalized TCR to the lysosome compartment after ligand engagement was reduced. These findings show that Cbl family proteins negatively regulate T cell activation by promoting clearance of engaged TCR from the cell surface, a process that is apparently essential for the termination of TCR signals.

L11 ANSWER 35 OF 81 MEDLINE on STN

ACCESSION NUMBER: 2003052597 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12561469
TITLE: The cloning and expression of a novel mouse gene mLPTS and its subcellular localization.
AUTHOR: Liao Cheng; Zhao Mu-Jun; Li Zai-Ping
CORPORATE SOURCE: State Key Laboratory of Molecular Biology, Institute of Biochemistry and Cell Biology, Shanghai, China.
SOURCE: Yi chuan xue bao = Acta genetica Sinica, (2002 Oct) 29 (10) 865-70.
Journal code: 7900784. ISSN: 0379-4172.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Chinese

FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF321817
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 20030204
Last Updated on STN: 20030624
Entered Medline: 20030623

TI The cloning and expression of a novel mouse gene mLPTS and its subcellular localization.

AB A novel mouse gene mLPTS was cloned by EST assembling, RT-PCR and DNA sequencing. The gene fragment for mLPTS is 1244 bp in length, encoding a protein of 332 amino acids. The amino acid sequence of mLPTS has 78% homologue with that of LPTS gene, which is a novel liver cancer-related gene identified through positional candidate cloning stratage by our laboratory. The expression of LPTS gene was **ubiquitous** in normal human tissues, whereas levels appeared to be significantly reduced, or sometime undetectable in HCC cells and neoplastic tissues, and it might be involved in the negative regulation of cell proliferation. The expression of mLPTS gene was found in all mouse tissues analyzed, same with that of LPTS gene in human. There was only one transcript for mLPTS gene in mouse tissues. The phylogenetic tree was constructed through the amino acids sequence analysis and the study of the sequence homologue among different species. Next, mLPTS gene was cloned into green fluorescent protein eukarytic expression vector and then transfected into CHO cell line. The green fluorescent was mostly limited in the nucleolus, showing that the gene products of mLPTS in eukaryocytes were located in the nucleolus.

L11 ANSWER 36 OF 81 MEDLINE on STN
ACCESSION NUMBER: 2003371814 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12905781
TITLE: TCR-mediated signaling.
AUTHOR: Yang Chun; Zhu Li-ping; Zhang Wei
CORPORATE SOURCE: Department of Clinical Medicine, PUMC, Beijing 100730, China.
SOURCE: Zhongguo yi xue ke xue yuan xue bao. Acta Academiae Medicinae Sinicae, (2002 Oct) 24 (5) 532-6. Ref: 8
Journal code: 8006230. ISSN: 1000-503X.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: Chinese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20030809
Last Updated on STN: 20040602
Entered Medline: 20040601

TI TCR-mediated signaling.

AB In recent years, major progress has been obtained in studying the early events in TCR-mediated signaling. c-Cbl has been found to be a negative regulatory factor of the tyrosine kinases in ZAP-70/SyK family. The studies on LAT, **SLP**-76, ItK and Vav have shown their roles in the signal transduction of Ras and phospholipase Cx1 to Ca2+. Micro-glycolipid raft also plays important role in T cell activation. This minireview shows a brief introduction to the process of TCR-mediated signaling.

L11 ANSWER 37 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 2002:339260 CAPLUS
DOCUMENT NUMBER: 137:135232
TITLE: Androgen receptor interactions with Oct-1 and Brn-1 are physically and functionally distinct
AUTHOR(S): Gonzalez, M. Ivelisse; Tovaglieri, Alessandra; Robins,

Diane M.
CORPORATE SOURCE: Department of Human Genetics, University of Michigan Medical School, Ann Arbor, MI, 48109-0618, USA
SOURCE: Molecular and Cellular Endocrinology (2002), 190(1-2), 39-49
CODEN: MCEND6; ISSN: 0303-7207
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Androgen receptor interactions with Oct-1 and Brn-1 are physically and functionally distinct

AB POU domain proteins interact pos. or neg. with steroid hormone receptors, depending on the precise array of these and other factors assembled on target gene promoters. Octamer transcription factor 1 (Oct-1), a **ubiquitous** POU factor, is implicated in androgen induction of the mouse sex-limited protein (**Slp**) gene based on protein-DNA interaction studies. However, direct evidence for a role of Oct-1 in the hormone response has been difficult to obtain. Brain 1 (Brn-1), another POU factor, is more tissue-specific, expressing in brain and also in kidney, which is a major site of **Slp** synthesis. We compared the interaction of the androgen receptor (AR) with Oct-1 and Brn-1 to reveal the more likely candidate for regulation of **Slp**. In transfection, addition of either Oct-1 or Brn-1 reduced AR activation, regardless of the presence of an octamer-like sequence in the enhancer, suggesting interference was indirect. However, when the octamer-like element was changed to a consensus octamer site, Brn-1, but not Oct-1, strongly enhanced androgen activation. This correlated with Brn-1's preference for the consensus octamer sequence in DNA binding assays. Direct interaction of AR with glutathione-S-transferase-(GST)-fused Oct-1 was DNA-dependent, while Brn-1-AR association was not. Chimeric Brn-1 and Oct-1 POU domains demonstrated that the DNA-dependent AR interaction relied on the origin of the POU homeodomain. However, in the context of full-length Brn-1 and Oct-1 chimeric proteins, the POU homedomain was not sufficient to confer the distinct behaviors of these factors in vivo, but instead revealed the importance of an N-terminal transactivation domain in Brn-1. These results demonstrate that functional interaction of Oct-1 and Brn-1 with AR is determined by the precise sequence of the octamer binding site, and by differential interaction of the POU factors with AR and other components of the transcriptional machinery.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 38 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:851435 CAPLUS
DOCUMENT NUMBER: 136:1570
TITLE: Compositions, kits, and methods for identification and modulation of T helper-1 and T helper-2 cells and diseases associated therewith
INVENTOR(S): Hanrahan, Catherine F.; Feldman, Marc; Trepicchio, William L.
PATENT ASSIGNEE(S): Genetics Institute, Inc., USA; Kennedy Institute of Rheumatology
SOURCE: PCT Int. Appl., 115 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001088199	A2	20011122	WO 2001-US16022	20010517
WO 2001088199	A3	20030206		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2409154	AA	20011122	CA 2001-2409154	20010517
US 2002039734	A1	20020404	US 2001-860655	20010517
EP 1299560	A2	20030409	EP 2001-933353	20010517

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.:	US 2000-205204P	P	20000518
	WO 2001-US16022	W	20010517

TI Compositions, kits, and methods for identification and modulation of T helper-1 and T helper-2 cells and diseases associated therewith

AB The invention relates to compns., kits and methods for identifying, detecting, and modulating the differentiation, growth, and/or maturation of Th1 or Th2 cells. The invention further relates to compns., kits, and methods for detecting, characterizing, preventing, and treating a Th1- or Th2-associated condition. A variety of markers are provided, wherein changes in the levels of expression of one or more of the markers is correlated with the presence of a Th1 or Th2 cell or Th1- or Th2-associated condition. Macrophage inhibitory factor (MIF) gene expression which is increased in both Th1-inducing and TH2-inducing condition is analyzed.

L11 ANSWER 39 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER:	2001:300915 CAPLUS
DOCUMENT NUMBER:	134:336654
TITLE:	Criteria for the identification of housekeeping genes and their use as internal standards in the measurement of levels of gene expression
INVENTOR(S):	Warrington, Janet A.; Mahadevappa, Mamatha; Nair, Archana
PATENT ASSIGNEE(S):	Affymetrix, Inc., USA
SOURCE:	PCT Int. Appl., 60 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001029267	A1	20010426	WO 2000-US29252	20001023

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6841348	B1	20050111	US 2000-693204	20001019
EP 1226278	A1	20020731	EP 2000-973795	20001023

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

US 2005053998	A1	20050310	US 2004-968652	20041018
PRIORITY APPLN. INFO.:			US 1999-161000P	P 19991021
			US 2000-693204	A1 20001019

WO 2000-US29252 W 20001023

TI Criteria for the identification of housekeeping genes and their use as internal standards in the measurement of levels of gene expression

AB This invention provides methods for discovering maintenance genes and for using maintenance genes. In one embodiment, the expression of at least three maintenance genes are measured and used as reference (or control) for comparing the expression of target genes in two or more biol. samples. Genes are identified as housekeeping genes by measuring their levels of expression relative to a large panel (.apprx.100 or more) known housekeeping genes in a number of tissues. If the relative level of expression of the gene does not vary outside a given range it is identified as a housekeeping gene.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 40 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 10

ACCESSION NUMBER: 2001:170264 CAPLUS

DOCUMENT NUMBER: 134:231996

TITLE: Oct-1 preferentially interacts with androgen receptor in a DNA-dependent manner that facilitates recruitment of SRC-1

AUTHOR(S): Gonzalez, M. Ivelisse; Robins, Diane M.

CORPORATE SOURCE: Department of Human Genetics and Cell and Molecular Biology Program, University of Michigan Medical School, Ann Arbor, MI, 48109-0618, USA

SOURCE: Journal of Biological Chemistry (2001), 276(9), 6420-6428
CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

TI Oct-1 preferentially interacts with androgen receptor in a DNA-dependent manner that facilitates recruitment of SRC-1

AB Gene regulation by steroid hormone receptors depends on the particular character of the DNA response element, the array of neighboring transcription factors, and recruitment of coactivators that interface with the transcriptional machinery. The authors are studying these complex interactions for the androgen-dependent enhancer of the mouse sex-limited protein (**Slp**) gene. This enhancer has, in addition to multiple androgen receptor (AR)-binding sites, a central region (FPIV) with a binding site for the **ubiquitous** transcription factor Oct-1 that appears crucial for hormonal regulation in vivo. To examine the role of Oct-1 in androgen-specific gene activation, the authors tested the interaction of Oct-1 with AR vs. glucocorticoid receptor (GR) in vivo and in vitro. Oct-1 coimmunopptd. from cell lysates with both AR and GR, but significant association with AR required both proteins to be DNA-bound. This was confirmed by sensitivity of the protein association to treatment with ethidium bromide or micrococcal nuclease. Addition of DNA to micrococcal nuclease-treated samples restored interaction, even when binding sites were on sep. DNA mols., suggesting association was due to direct protein-protein interaction and not indirect tethering via the DNA. AR/GR chimeras revealed that interaction of the N and C termini of AR was required to communicate the DNA-bound state that enhances interaction with Oct-1. Protease digestion assays of hormone-bound receptors revealed further conformational changes in the ligand binding domain of AR, but not GR, upon DNA binding. Furthermore, these conformational changes led to increased interaction with the coactivator SRC-1, via the NID 4 domain, suggesting DNA binding facilitates recruitment of SRC-1 by the AR-Oct-1 complex. Altogether, these results suggest that the precise arrangement of binding sites in the **Slp** enhancer ensures proper hormonal response by imposing differential interactions between receptors and Oct-1, which in turn contributes to SRC-1 recruitment to the promoter.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 41 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:401494 CAPLUS
DOCUMENT NUMBER: 135:150798
TITLE: Expression profiling and identification of novel genes in hepatocellular carcinomas
AUTHOR(S): Graveel, Carrie R.; Jatkoe, Tim; Madore, Steven J.; Holt, Alison L.; Farnham, Peggy J.
CORPORATE SOURCE: McArdle Laboratory for Cancer Research, University of Wisconsin-Madison, Madison, WI, 53706, USA
SOURCE: Oncogene (2001), 20(21), 2704-2712
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Expression profiling and identification of novel genes in hepatocellular carcinomas
AB Liver cancer is the fifth most common cancer worldwide and unlike certain other cancers, such as colon cancer, a mutational model has not yet been developed. The authors have performed gene expression profiling of normal and neoplastic livers in C3H/HeJ mice treated with diethylnitrosamine. Using oligonucleotide microarrays, the authors compared gene expression in liver tumors to three different states of the normal liver: quiescent adult, regenerating adult, and newborn. Although each comparison revealed hundreds of differentially expressed genes, only 22 genes were deregulated in the tumors in all three comparisons. Three of these genes were examined in human hepatocellular carcinomas and were upregulated. As a second method of anal., the authors used Representational Difference Anal. (RDA) to clone mRNA fragments differentially expressed in liver tumors vs. regenerating livers. The authors cloned several novel mRNAs that are differentially regulated in murine liver tumors. Here the authors report the sequence of a novel cDNA whose expression is upregulated in both murine and human hepatocellular carcinomas. The authors' results suggest that DEN-treated mice provide an excellent model for human hepatocellular carcinomas.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 42 OF 81 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:526809 BIOSIS
DOCUMENT NUMBER: PREV200100526809
TITLE: A conserved **ubiquitin** ligase of the nuclear envelope/endoplasmic reticulum that functions in both ER-associated and Matalpha2 repressor degradation.
AUTHOR(S): Swanson, Robert; Locher, Martin; Hochstrasser, Mark [Reprint author]
CORPORATE SOURCE: Department of Molecular Biophysics and Biochemistry, Yale University, New Haven, CT, 06520, USA
mark.hochstrasser@yale.edu
SOURCE: Genes and Development, (October 15, 2001) Vol. 15, No. 20, pp. 2660-2674. print.
CODEN: GEDEEP. ISSN: 0890-9369.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
TI A conserved **ubiquitin** ligase of the nuclear envelope/endoplasmic reticulum that functions in both ER-associated and Matalpha2 repressor degradation.
AB Substrate discrimination in the **ubiquitin**-proteasome system is

believed to be dictated by specific combinations of **ubiquitin**-protein ligases (E3s) and **ubiquitin**-conjugating enzymes (E2s). Here we identify Doa10/Ssm4 as a yeast E3 that is embedded in the endoplasmic reticulum (ER)/nuclear envelope yet can target the soluble transcription factor Matalpha2. Doa10 contains an unusual RING finger, which has **ubiquitin**-ligase activity in vitro and is essential in vivo for degradation of alpha2 via its Deg1 degradation signal. Doa10 functions with two E2s, Ubc6 and Ubc7, to **ubiquitinate** Deg1-bearing substrates, and it is also required for the degradation of at least one ER membrane protein. Interestingly, different short-lived ER proteins show distinct requirements for Doa10 and another ER-localized E3, Hrd1. Nevertheless, the two E3s overlap in function: A doa10DELTA hrd1DELTA mutant is far more sensitive to cadmium relative to either single mutant and displays strong constitutive induction of the unfolded protein response; this suggests a role for both E3s in eliminating aberrant ER proteins. The likely human ortholog of DOA10 is in the **cir**-du-chat syndrome critical region on chromosome 5p, suggesting that defective **ubiquitin** ligation might contribute to this common genetic disorder.

L11 ANSWER 43 OF 81 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:258370 BIOSIS
DOCUMENT NUMBER: PREV200100258370
TITLE: The Grb2-related adaptor protein GrpL/Gads associates with essential B cell signaling molecules.
AUTHOR(S): Yankee, Thomas M. [Reprint author]; Draves, Kevin E. [Reprint author]; Ewings, Maria K. [Reprint author]; Clark, Edward A. [Reprint author]
CORPORATE SOURCE: University of Washington, 1959 NE Pacific St, Seattle, WA, 98195, USA
SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1041. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001. Orlando, Florida, USA. March 31-April 04, 2001.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 May 2001
Last Updated on STN: 19 Feb 2002

TI The Grb2-related adaptor protein GrpL/Gads associates with essential B cell signaling molecules.

AB Adaptor proteins play a key role in the selection of pathways stimulated by the engagement of antigen receptors. We have characterized the role of the Grb2 family member GrpL (also known as Gads/Mona/Grf40/GRID) in signaling through the B cell antigen receptor (BCR). We first defined signaling proteins that interact with GrpL. Untreated or anti-IgM-treated human MP-1 B cells were immunoprecipitated with a GrpL monoclonal antibody and subjected to immune complex kinase assays. Four heavily phosphorylated proteins with the approximate molecular weights of 35-40 kD, 75-80 kD, 95-100 kD, and 110-120 kD were present in untreated cells. Following BCR ligation, the same four proteins were present in the complex, but the kinase activity associated with GrpL was enhanced. The bands containing these four molecules were excised, and the proteins were eluted from the gel and re-immunoprecipitated with antibodies against potential binding partners. These experiments revealed that these proteins are GrpL itself, the **SLP**-76 adaptor, the HPK1 kinase, and the c-Cbl **ubiquitin** ligase. In addition, these proteins co-immunoprecipitated with GrpL, as detected by Western blotting. Also, 14-3-3 proteins inducibly interact with GrpL following BCR ligation. To test the role of the interaction between GrpL and **SLP**-76, the

GrpL-negative human B cell line BJAB was transfected with cDNA encoding GrpL, **SLP**-76, or both. Like in T cells, GrpL and **SLP**-76 synergize to augment antigen receptor-mediated NF-AT activity. We are testing the hypothesis that HPK1 phosphorylates GrpL and creates a docking site for 14-3-3 proteins. The phosphorylation of GrpL, then, may regulate the nature and duration of the interactions between GrpL and other signaling molecules.

L11 ANSWER 44 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 11
ACCESSION NUMBER: 2001:863087 CAPLUS
DOCUMENT NUMBER: 136:117174
TITLE: Microarray analysis of eosinophils reveals a number of candidate survival and apoptosis genes
AUTHOR(S): Temple, Roger; Allen, Elizabeth; Fordham, Jeremy; Phipps, Simon; Schneider, Hans-Christoph; Lindauer, Klaus; Hayes, Ian; Lockey, Jacqui; Pollock, Kenny; Jupp, Ray
CORPORATE SOURCE: Aventis Pharmaceuticals, Inc., Bridgewater, NJ, 08807, USA
SOURCE: American Journal of Respiratory Cell and Molecular Biology (2001), 25(4), 425-433
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Thoracic Society
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Microarray analysis of eosinophils reveals a number of candidate survival and apoptosis genes
AB The increase in eosinophils at the site of antigen challenge has been used as evidence to suggest that this cell type plays a role in the pathophysiol. of asthma. Aberrant production of several different cytokines, particularly interleukin (IL)-5, has been shown to result in eosinophilia. IL-5 influences the development and maturation of eosinophils in a number of different ways. Of note is the ability of IL-5 to act as a survival factor for eosinophils specifically inhibiting apoptosis. The precise mechanism by which IL-5 exerts its effect remains obscure. We used microarray technologies to investigate the changes in the mRNA expression profile of eosinophils after treatment with IL-5. Using the Affymetrix Hu6800 chip, a total of 80 genes were observed to be regulated by 2-fold or greater. Many of the genes previously identified as regulated by IL-5 were regulated in our microarray expts. Of the 73 genes found to be upregulated, many were shown to play a role in adhesion, migration, activation, or survival of eosinophils or hematopoietic cells, whereas the function of others was unknown. To facilitate the identification of genes that govern the apoptosis and survivability of eosinophils, we used an alternative cellular model, TF1.8 cells, whose survival was also dependent on IL-5. Comparison of these models identified four genes, Pim-1, DSP-5 (hVH3, B23), CD24, and **SLP**-76, whose regulation was similarly coordinated in both systems. Identification of Pim-1 and **SLP**-76 as regulated by IL-5 led us to suggest a direct role for these proteins in the IL-5 signaling pathway in eosinophils. The tissue distribution of these genes demonstrated that Pim-1 and **SLP**-76 were relatively restricted to the eosinophil compared with their expression in brain, bone marrow, kidney, liver, and lung. By contrast, DSP-5 and CD24 were confirmed as **ubiquitous** in their expression by microarray.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 45 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:730469 CAPLUS
DOCUMENT NUMBER: 136:84625
TITLE: Biological insights into TCR$\gamma\delta$+ and TCR$\alpha\beta$+ intraepithelial lymphocytes provided by serial analysis of gene expression (SAGE)

AUTHOR(S): Shires, John; Theodoridis, Efstathios; Hayday, Adrian C.
CORPORATE SOURCE: Peter Gorer Department of Immunobiology Guy's, King's, Medical School King's College, University of London, London, SE1 9RT, UK
SOURCE: Immunity (2001), 15(3), 419-434
CODEN: IUNIEH; ISSN: 1074-7613
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Biological insights into TCRγδ+ and TCRαβ+ intraepithelial lymphocytes provided by serial analysis of gene expression (SAGE)

AB Intraepithelial lymphocytes (IELs) are abundant, evolutionarily conserved T cells, commonly enriched in T cell receptor (TCR)γδ expression. However, their primary functional potential and constitutive activation state are incompletely understood. To address this, serial anal. of gene expression (SAGE) was applied to murine TCRγδ+ and TCRαβ+ intestinal IELs directly ex vivo, identifying 15,574 unique transcripts that collectively portray an "activated yet resting," Th1-skewed, cytolytic, and immunoregulatory phenotype applicable to multiple subsets of gut IELs. Expression of granzymes, Fas ligand, RANTES, prothymosin β4, junB, RGS1, Btg1, and related mols. is high, whereas expression of conventional cytokines and high-affinity cytokine receptors is low. Differentially expressed genes readily identify heterogeneity among TCRαβ+ IELs, whereas differences between resident TCRγδ+ IELs and TCRαβ+ IELs are less obvious.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 46 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:623311 SCISEARCH
THE GENUINE ARTICLE: 457DZ
TITLE: Complete sequence, genomic organization, and chromosomal localization of the human gene encoding the SHP2-interacting transmembrane adaptor protein (SIT)
AUTHOR: Hubener C; Mincheva A; Lichter P; Schraven B; Bruyns E (Reprint)
CORPORATE SOURCE: LYNX Therapeut GMBH, Neuenheimer Feld 515, D-69120 Heidelberg, Germany (Reprint); Univ Heidelberg, Immunomodulat Lab, Inst Immunol, D-69120 Heidelberg, Germany; German Canc Res Ctr, Div Org Complex Genomes, D-69120 Heidelberg, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: IMMUNOGENETICS, (MAY-JUN 2001) Vol. 53, No. 4, pp. 337-341

Publisher: SPRINGER-VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010 USA.
ISSN: 0093-7711.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 19

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI Complete sequence, genomic organization, and chromosomal localization of the human gene encoding the SHP2-interacting transmembrane adaptor protein (SIT)

AB Engagement of the T-cell antigen receptor (TCR) by monoclonal antibodies or antigen/MHC complexes ultimately induces cellular responses such as proliferation, differentiation., apoptosis, or anergy. Signal propagation from the TCR to the nucleus depends on a variety of transmembrane and intracellular proteins. Tyrosine phosphorylations of

these molecules by protein tyrosine kinases (PTKs) of the src and syk families are among the earliest events occurring after TCR engagement and are prerequisites for the coupling of the TCR to intracellular signalling pathways such as the Ras/Raf/MAPK- or the calcineurin/NF-AT pathways. In addition, TCR-mediated tyrosine phosphorylation plays a key role in the translocation of intracellular signalling molecules (e.g., Grb2, SHP2, **SLP**-76) from the cytosol to the inner leaflet of the plasma membrane, a process partially mediated by a recently described group of signalling molecules termed transmembrane adaptor proteins (Schraven et al. 1999).

We have recently reported the identification of one of the transmembrane adaptor proteins named SHP2-interacting transmembrane adaptor protein (SIT) (Marie-Cardine et al. 1999). Similar to the other transmembrane adaptor proteins (LAT, TRIM, PAG/cbp), SIT consists of a short extracellular domain and a comparatively long cytoplasmic tail containing several tyrosine-based signalling motifs which become rapidly phosphorylated by PTKs upon T-cell activation and then serve as docking sites for SH2 domain-containing intracellular signalling molecules. SIT inducibly interacts with the SH2-containing tyrosine phosphatase 2 (SHP2) via an immunoreceptor tyrosine-based inhibition motif as well as with the adaptor protein Grb2 (growth factor receptor binding protein 2) via two consensus YxN motifs. In addition. SIT seems to inhibit the induction of the transcriptional activity of the nuclear factor of T cells (NF-AT) upon TCR engagement. This function is mediated via recruitment of a negative regulatory effector molecule, most likely the tyrosine kinase p50csk (Pfrepper et al., 2001).

In the present study, we isolated the SIT gene from a human P-1-derived artificial chromosome (PAC) library and determined the exon-intron junctions. We additionally analyzed a 2.5-kb 5 ' upstream region and identified a TATA-less sequence containing potential binding sites for both **ubiquitous** and lymphoid-specific transcription factors (Sp-1, ETF, NF-kappaB, Ikaros, GATA). We demonstrated promoter activity of this fragment by transient transfection analysis and mapped the SIT gene to its genomic locus 9p12-13 by fluorescence in situ hybridization (FISH).

L11 ANSWER 47 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:878426 CAPLUS
DOCUMENT NUMBER: 134:191523
TITLE: The transcriptional responses of respiratory epithelial cells to Bordetella pertussis reveal host defensive and pathogen counter-defensive strategies
AUTHOR(S): Belcher, Christopher E.; Drenkow, Jorg; Kehoe, Bettina; Gingeras, Thomas R.; McNamara, Nancy; Lemjabbar, Hassan; Basbaum, Carol; Relman, David A.
CORPORATE SOURCE: Departments of Pediatrics, Stanford University, Stanford, CA, 94305, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(25), 13847-13852
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

TI The transcriptional responses of respiratory epithelial cells to Bordetella pertussis reveal host defensive and pathogen counter-defensive strategies

AB Bordetella pertussis, the causative agent of whooping cough, has many well-studied virulence factors and a characteristic clin. presentation. Despite this information, it is not clear how B. pertussis interaction with host cells leads to disease. In this study, we examined the interaction of B. pertussis with a human bronchial epithelial cell line (BEAS-2B) and measured host transcriptional profiles by using high-d. DNA microarrays. The early transcriptional response to this pathogen is dominated by altered expression of cytokines, DNA-binding proteins, and

NFκB-regulated genes. This previously unrecognized response to B. pertussis was modified in similar but nonidentical fashions by the antiinflammatory agents dexamethasone and sodium salicylate. Cytokine protein expression was confirmed, as was neutrophil chemoattraction. We show that B. pertussis induces mucin gene transcription by BEAS-2B cells then counters this defense by using mucin as a binding substrate. A set of genes is described for which the catalytic activity of pertussis toxin is both necessary and sufficient to regulate transcription. Host genomic transcriptional profiling, in combination with functional assays to evaluate subsequent biol. events, provides insight into the complex interaction of host and pathogen.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 48 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:236540 CAPLUS
DOCUMENT NUMBER: 133:87817
TITLE: Mitotic misregulation and human aging
AUTHOR(S): Ly, Danith H.; Lockhart, David J.; Lerner, Richard A.; Schultz, Peter G.
CORPORATE SOURCE: Department of Chemistry and the Skaggs Institute for Chemical Biology, The Scripps Research Institute, La Jolla, CA, 92037, USA
SOURCE: Science (Washington, D. C.) (2000), 287(5462), 2486-2492
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Mitotic misregulation and human aging
AB MRNA levels were measured in actively dividing fibroblasts isolated from young, middle-age, and old-age humans, and humans with progeria, a rare genetic disorder characterized by accelerated aging. Genes whose expression was associated with age-related phenotypes and diseases were identified. The data suggest that an underlying mechanism of the aging process involves increasing errors in the mitotic machinery of dividing cells in the postreproductive stage of life. This dysfunction may lead to chromosomal pathologies that result in misregulation of genes involved in the aging process.
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 49 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:176289 CAPLUS
DOCUMENT NUMBER: 133:250839
TITLE: Profound misregulation of muscle-specific gene expression in facioscapulohumeral muscular dystrophy. [Erratum to document cited in CA132:48549]
AUTHOR(S): Tupler, Rossella; Perini, Giovanni; Pellegrino, Maria Antonietta; Green, Michael R.
CORPORATE SOURCE: Howard Hughes Medical Institute, Program in Molecular Medicine, Massachusetts Medical School, Worce, MA, 01605, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(5), 2397
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Profound misregulation of muscle-specific gene expression in facioscapulohumeral muscular dystrophy. [Erratum to document cited in CA132:48549]
AB GenBank accession nos. AW0055750-69 should read AW055750-69.

L11 ANSWER 50 OF 81 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 12

ACCESSION NUMBER: 2000:94415 BIOSIS
DOCUMENT NUMBER: PREV200000094415
TITLE: Regional fine mapping of **HMG17** to chromosomal band 1p35.
AUTHOR(S): Kazmierczak, Bernd; Dal Cin, Paola; Rogalla, Piere; Van den Berghe, Herman; Bullerdiek, Joern [Reprint author]
CORPORATE SOURCE: Center for Human Genetics, University of Bremen, ZHG, Leobenerstr., D-28359, Bremen, Germany
SOURCE: Cancer Genetics and Cytogenetics, (Jan. 15, 2000) Vol. 116, No. 2, pp. 164-165. print.
CODEN: CGCYDF. ISSN: 0165-4608.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2000
Last Updated on STN: 3 Jan 2002

TI Regional fine mapping of **HMG17** to chromosomal band 1p35.

AB **HMG17**, a member of the high-mobility group of proteins, is **ubiquitously** expressed in higher eukaryotes. This protein has been shown to enhance transcriptional activity of many other genes. Recently, the **HMG17** gene has been mapped to chromosomal band 1p36.1. Because this region is frequently involved in chromosomal aberrations of various human neoplasms, two PAC clones containing an **HMG17** sequence were isolated. By fluorescence in situ hybridization (FISH) on prometaphase spreads, **HMG17** was mapped to chromosomal band 1p35.

L11 ANSWER 51 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1999:728918 CAPLUS
DOCUMENT NUMBER: 132:48549
TITLE: Profound misregulation of muscle-specific gene expression in facioscapulohumeral muscular dystrophy
AUTHOR(S): Tupler, Rossella; Perini, Giovanni; Pellegrino, Maria Antonietta; Green, Michael R.
CORPORATE SOURCE: Howard Hughes Medical Institute, Program in Molecular Medicine, University of Massachusetts Medical School, Worce, MA, 01605, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1999), 96(22), 12650-12654
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Profound misregulation of muscle-specific gene expression in facioscapulohumeral muscular dystrophy

AB Facioscapulohumeral muscular dystrophy (FSHD) is a neuromuscular disorder characterized by an insidious onset and progressive course. The disease has a frequency of about 1 in 20,000 and is transmitted in an autosomal dominant fashion with almost complete penetrance. Deletion of an integral number of tandemly arrayed 3.3-kb repeat units (D4Z4) on chromosome 4q35 is associated with FSHD but otherwise the mol. basis of the disease and its pathophysiol. remain obscure. Comparison of mRNA populations between appropriate cell types can facilitate identification of genes relevant to a particular biol. or pathol. process. In this report, the authors have compared mRNA populations of FSHD and normal muscle. Unexpectedly, the dystrophic muscle displayed profound alterations in gene expression characterized by severe underexpression or overexpression of specific mRNAs. Intriguingly, many of the deregulated mRNAs are muscle specific. The authors' results suggest that a global misregulation of gene expression is the underlying basis for FSHD, distinguishing it from other forms of muscular dystrophy. The exptl. approach used here is applicable

to any genetic disorder whose pathogenic mechanism is incompletely understood.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 52 OF 81 MEDLINE on STN
ACCESSION NUMBER: 1999288070 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10358158
TITLE: Perturbed regulation of ZAP-70 and sustained tyrosine phosphorylation of LAT and **SLP**-76 in c-Cbl-deficient thymocytes.
AUTHOR: Thien C B; Bowtell D D; Langdon W Y
CORPORATE SOURCE: Department of Pathology, University of Western Australia, Queen Elizabeth II Medical Center, Nedlands.
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (1999 Jun 15) 162 (12) 7133-9.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990630

TI Perturbed regulation of ZAP-70 and sustained tyrosine phosphorylation of LAT and **SLP**-76 in c-Cbl-deficient thymocytes.

AB Recent studies indicate that c-Cbl and its oncogenic variants can modulate the activity of protein tyrosine kinases. This finding is supported by studies showing that c-Cbl interacts directly with a negative regulatory tyrosine in ZAP-70, and that the levels of tyrosine-phosphorylated ZAP-70 and numerous other proteins are increased in TCR-stimulated thymocytes from c-Cbl-deficient mice. Here, we demonstrate that this enhanced phosphorylation of ZAP-70 and that of two substrates, LAT and **SLP** -76, is not due to altered protein levels but is the consequence of two separate events. First, we find increased expression of tyrosine-phosphorylated TCRzeta chain in c-Cbl-deficient thymocytes, which results in a higher level of zeta-chain-associated ZAP-70 that is initially accessible for activation. Thus, more ZAP-70 is activated and more of its substrates (LAT and **SLP**-76) become tyrosine-phosphorylated after TCR stimulation. However, an additional mechanism of ZAP-70 regulation is evident at a later time poststimulation. At this time, ZAP-70 from both normal and c-Cbl-/- thymocytes becomes hyperphosphorylated; however, only in normal thymocytes does this correlate with ZAP-70 down-regulation and a diminished ability to phosphorylate LAT and **SLP**-76. In contrast, c-Cbl-deficient thymocytes display altered phosphorylation kinetics, for which LAT phosphorylation is increased and **SLP**-76 phosphorylation is sustained. Thus, the ability to down-regulate the phosphorylation of two ZAP-70 substrates is impaired in c-Cbl-/- thymocytes. These findings provide evidence that c-Cbl is involved in the negative regulation of the phosphorylation of LAT and **SLP**-76 by ZAP-70.

L11 ANSWER 53 OF 81 MEDLINE on STN
ACCESSION NUMBER: 1999244078 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10229072
TITLE: Association of Nck with tyrosine-phosphorylated **SLP** -76 in activated T lymphocytes.
AUTHOR: Wunderlich L; Farago A; Downward J; Buday L
CORPORATE SOURCE: Department of Medical Chemistry, Semmelweis University Medical School, Budapest, Hungary.
SOURCE: European journal of immunology, (1999 Apr) 29 (4) 1068-75.
Journal code: 1273201. ISSN: 0014-2980.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990525
Last Updated on STN: 19990525
Entered Medline: 19990513

TI Association of Nck with tyrosine-phosphorylated **SLP**-76 in activated T lymphocytes.

AB The Nck adaptor protein links tyrosine kinases or their substrates to proteins containing proline-rich motifs. Here we show that in activated T cells two tyrosine phosphoproteins of 75 and 120 kDa are co-immunoprecipitated with polyclonal antibodies against Nck. Analysis of Nck immunoprecipitates with various candidate antibodies revealed that the 75-kDa tyrosine phosphoprotein is the SH2 domain-containing leukocyte protein referred to as **SLP**-76. In vitro experiments show that the interaction between Nck and **SLP**-76 is mediated via the Nck SH2 domain. Using specific phosphopeptides corresponding to the major tyrosine phosphorylation sites of **SLP**-76, it was found that Y113 and Y128 phosphopeptides could compete binding of **SLP**-76 to the SH2 domain of Nck. In addition, the 120-kDa tyrosine phosphoprotein was recognized by an antibody raised against Cbl, a proto-oncogene product that has been previously found to be associated with Nck. These results suggest that the Nck adaptor protein interacts with key signaling molecules and may play an important role in activation of T lymphocytes.

L11 ANSWER 54 OF 81 MEDLINE on STN
ACCESSION NUMBER: 1999219276 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10204582
TITLE: Cbl functions downstream of Src kinases in Fc gamma RI signaling in primary human macrophages.
AUTHOR: Erdreich-Epstein A; Liu M; Kant A M; Izadi K D; Nolta J A; Durden D L
CORPORATE SOURCE: Neil Bogart Memorial Laboratories, Department of Pediatrics, Childrens Hospital Los Angeles Research Institute, University of Southern California School of Medicine, USA.
CONTRACT NUMBER: RO1 CA7563701 (NCI)
RO1 DK53041 (NIDDK)
SOURCE: Journal of leukocyte biology, (1999 Apr) 65 (4) 523-34.
Journal code: 8405628. ISSN: 0741-5400.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990511
Last Updated on STN: 20000303
Entered Medline: 19990429

TI Cbl functions downstream of Src kinases in Fc gamma RI signaling in primary human macrophages.

AB Cbl is a cytosolic protein that is rapidly tyrosine phosphorylated in response to Fc receptor activation and binds to the adaptor proteins Grb2, CrkL, and Nck. A few reports describe Cbl interactions in primary human hematopoietic cells. We show evidence that Cbl participates in signaling initiated by Fc gammaRI receptor cross-linking in human primary macrophages, and functions downstream of Src family kinases in this pathway. Fc gammaRI stimulation in human macrophages was associated with rapid and transient tyrosine phosphorylation of the Cbl adaptor protein. Immunoprecipitated Cbl was complexed with several tyrosine phosphorylated proteins, the most prominent of which was a 38-kDa band identified as the CrkL adaptor protein. CrkL associated with tyrosine-phosphorylated Cbl

and itself became tyrosine phosphorylated after Fc gammaRI cross-linking. **SLP**-76, a recently cloned Grb2-associated protein, was strongly tyrosine phosphorylated after Fc gammaRI stimulation and was associated with both Cbl and Grb2. Grb2 and Cbl binding to **SLP**-76 were inducible after Fc gammaRI stimulation of the macrophages. Nck was inducibly bound to Cbl after Fc gammaRI stimulation, whereas Grb2 was constitutively associated with it. Shc was also inducibly tyrosine phosphorylated and bound to Grb2 after Fc gammaRI stimulation of the macrophages. PP1, a specific inhibitor of Src kinases, inhibited the Fc gammaRI-induced respiratory burst, as well as the tyrosine phosphorylation of Cbl and its inducible association with CrkL. These results suggest a fundamental role for the tyrosine phosphorylation of Cbl, CrkL, **SLP**-76, and Shc and the association of Cbl with CrkL, **SLP** -76, and Nck in Fc gammaRI signaling in human macrophages. Experiments performed with PP1, the specific Src kinase inhibitor, demonstrate the first evidence that Cbl and the Cbl-Crkl interaction are downstream targets for myeloid Src kinases required for the activation of myeloid NADPH oxidase activity.

L11 ANSWER 55 OF 81 MEDLINE on STN
ACCESSION NUMBER: 1999306920 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10375551
TITLE: T cell antigen-receptor signal transduction.
AUTHOR: van Leeuwen J E; Samelson L E
CORPORATE SOURCE: National Cancer Institute, Building 37, Room 1E24, Laboratory of Cellular and Molecular Biology, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD 20892, USA.. vanleeuj@box-v.nih.gov
SOURCE: Current opinion in immunology, (1999 Jun) 11 (3) 242-8. Ref: 81
Journal code: 8900118. ISSN: 0952-7915.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990910
Last Updated on STN: 20000303
Entered Medline: 19990820
TI T cell antigen-receptor signal transduction.
AB During the past year, major progress has been made in understanding proximal TCR signal-transduction events. Cbl has been identified as a negative regulator of kinases from the ZAP-70/Syk family. Studies on LAT, **SLP**-76, Itk and Vav have revealed their role in the activation of Ras and phospholipase-Cgamma1-Ca2+ signalling pathways. TCR-induced cytoskeletal changes involve signalling through **SLP**-76-Vav-Nck to activate effectors of the Rho-family of GTPases. Finally, glycolipid-enriched microdomains play a crucial role in T cell activation.

L11 ANSWER 56 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:806874 CAPLUS
DOCUMENT NUMBER: 133:54285
TITLE: Regional Fine Mapping of **HMG17** to Chromosomal Band 1p35
AUTHOR(S): Kazmierczak, B.; Dal Cin, P.; Rogalla, P.; Van den Berghe, H.; Bullerdiek, J.
CORPORATE SOURCE: Center for Human Genetics and Genetic Counseling, University of Bremen, Bremen, Germany
SOURCE: Cancer Genetics and Cytogenetics (1999), Volume Date 2000, 116(2), 164-165
CODEN: CGCYDF; ISSN: 0165-4608

PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Regional Fine Mapping of **HMG17** to Chromosomal Band 1p35
AB **HMG17**, a member of the high-mobility group of proteins, is **ubiquitously** expressed in higher eukaryotes. This protein has been shown to enhance transcriptional activity of many other genes. Recently, the **HMG17** gene has been mapped to chromosomal band 1p36.1. Because this region is frequently involved in chromosomal aberrations of various human neoplasms, two PAC clones containing an **HMG17** sequence were isolated. By fluorescence in situ hybridization (FISH) on prometaphase spreads, **HMG17** was mapped to chromosomal band 1p35.
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 57 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:647053 CAPLUS
DOCUMENT NUMBER: 134:204651
TITLE: Reconstitution of high mobility group 14/17 proteins into nucleosomes and chromatin
AUTHOR(S): Postnikov, Yuri V.; Bustin, Michael
CORPORATE SOURCE: Protein Section, Division of Basic Sciences, National Cancer Institute, National Institutes of Health, Bethesda, MD, 20892, USA
SOURCE: Methods in Enzymology (1999), 304(Chromatin), 133-155
CODEN: MENZAU; ISSN: 0076-6879
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Reconstitution of high mobility group 14/17 proteins into nucleosomes and chromatin
AB High mobility group (HMG) proteins are a **ubiquitous** and heterogeneous class of nonhistones, which serve as architectural elements that modify the structure of DNA and chromatin, thereby facilitating a variety of DNA-related activities in the nucleus of the cell. The HMG14/17 subgroup is the only class of nuclear proteins known to bind specifically to the 146-bp core particle. These proteins bind to the nucleosome core cooperatively and form homodimeric complexes containing either two mols. of HMG14 or two mols. of **HMG17**. Both HMG14 and **HMG17** contact the nucleosomal DNA 25 bp from the end of the core particle and in the two major grooves flanking the nucleosomal dyad axis. Addnl. specific contacts are made with the amino termini of the core histones. The proteins stimulate various DNA-dependent activities such as transcription and replication, but only in the context of chromatin. Enhancement of the DNA-dependent activities is associated with a decompaction, or unfolding, of the higher order chromatin structure. These findings indicate that HMG14/17 proteins are chromatin-specific architectural elements that stimulate DNA-dependent activities by unfolding the higher order chromatin structure and facilitating access to the underlying DNA sequence. Various approaches useful for studies on the structure and function of HMG14/17 proteins in chromatin, are described. These approaches include studies on the binding of HMG14/17 to chromatin subunits, on their reconstitution into in vitro assembled chromatin, and on their in situ organization in cellular chromatin. The methods described may be suitable for studies on other nonhistone proteins. (c) 1999 Academic Press.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 58 OF 81 MEDLINE on STN
ACCESSION NUMBER: 1998384233 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9716598

TITLE: **SLP**-76-Cbl-Grb2-Shc interactions in FcgammaRI signaling.
AUTHOR: Chu J; Liu Y; Koretzky G A; Durden D L
CORPORATE SOURCE: Neil Bogart Memorial Laboratories, Division of Hematology-Oncology, Childrens Hospital Los Angeles, Los Angeles, CA, USA.
CONTRACT NUMBER: R01CA75637-01 (NCI)
R01GM53256 (NIGMS)
SOURCE: Blood, (1998 Sep 1) 92 (5) 1697-706.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19980917
Last Updated on STN: 19980917
Entered Medline: 19980910

TI **SLP**-76-Cbl-Grb2-Shc interactions in FcgammaRI signaling.
AB **SLP**-76 and Cbl are complex adapter proteins that have the capacity to bind to smaller adapter proteins, such as Grb2, which subsequently binds the nucleotide exchange protein Sos in the transmission of intracellular signals. **SLP**-76, Cbl, Shc, and Grb2 have been implicated in immunoreceptor tyrosine-based activation motif (ITAM) signaling, leading to activation of Ras. However, their mechanism of action has not been determined. To date, there have been no reports of **SLP**-76 involvement in FcgammaRI-receptor signaling and no data exist for an interaction between Cbl, Shc, and **SLP**-76 in vivo. We provide evidence that **SLP**-76, Cbl, and Shc are tyrosine phosphorylated on FcgammaRI-receptor stimulation and are associated with the adapter protein Grb2 in gamma-interferon-differentiated U937 cells (U937IF). The interactions between **SLP**-76 and Cbl and **SLP**-76 and Grb2 are present in resting U937IF cells. However, the interaction between **SLP**-76 and Grb2 becomes augmented twofold on FcgammaRI-receptor aggregation. Our results provide the first evidence for a phosphorylation-dependent interaction between **SLP**-76 and Shc, induced at least 10-fold on FcgammaRI receptor stimulation. Our data indicate that a significant portion of a multimolecular complex containing Cbl, **SLP**-76, Shc, and Grb2 is distinct from a trimolecular complex containing the Ras guanine nucleotide exchanger Sos, Shc, and Grb2. FcgammaRI-induced tyrosine phosphorylation of **SLP**-76, Cbl, Shc, and the highly induced **SLP**-76-Shc interaction provide the first evidence that **SLP**-76 and Cbl are involved in FcgammaRI signaling and suggest a functional significance for these interactions in FcgammaRI signal relay in the control of Ras in myeloid cells.

L11 ANSWER 59 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:744537 SCISEARCH
THE GENUINE ARTICLE: 122AR
TITLE: SKAP-HOM, a novel adaptor protein homologous to the FYN-associated protein SKAP55
AUTHOR: MarieCardine A (Reprint); Verhagen A M; Eckerskorn C; Schraven B
CORPORATE SOURCE: UNIV HEIDELBERG, INST IMMUNOL, NEUENHEIMER FELD 305, D-69120 HEIDELBERG, GERMANY (Reprint); MAX PLANCK INST BIOCHEM, D-82152 MARTINSRIED, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: FEBS LETTERS, (11 SEP 1998) Vol. 435, No. 1, pp. 55-60.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0014-5793.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 14

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI SKAP-HOM, a novel adaptor protein homologous to the FYN-associated protein SKAP55

AB A recombinant GST-Fyn-SH2 domain was used to purify proteins from lysates of pervanadate treated EL4 cells. N-terminal sequencing and molecular cloning of one of the purified polypeptides resulted in the identification of a novel adaptor protein that shares strong structural homology to the recently cloned Fyn-associated adaptor protein SKAP55. This protein was termed SKAP-HOM ((SKAP) under bar 55 homologue). Despite their striking homology, SKAP55 and SKAP-HOM have distinct characteristics. Thus, unlike SKAP55, which is exclusively expressed in T lymphocytes, SKAP-HOM expression is **ubiquitous**. Furthermore, while SKAP55 is constitutively tyrosine phosphorylated in resting human T cells, SKAP-HOM is expressed as a non-phosphorylated protein in the absence of external stimulus but becomes phsphorylated following T cell activation. In addition, SKAP-HOM does not associate with p59(fyn) in T cells although it represents a specific substrate for the kinase in COS cells. Finally, we demonstrate that, as previously shown for SKAP55, SKAP-HOM interacts with the recently identified polypeptide SLAP-130. (C) 1998 Federation of European Biochemical Societies.

L11 ANSWER 60 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 97:533050 SCISEARCH
THE GENUINE ARTICLE: XJ994
TITLE: A climatology of coastal ridging over south-eastern Australia
AUTHOR: Speer M S (Reprint); Leslie L M
CORPORATE SOURCE: BUR METEOROL, REG FORECASTING CTR, POB 413, DARLINGHURST, NSW 2000, AUSTRALIA (Reprint); UNIV NEW S WALES, SCH MATH, SYDNEY, NSW, AUSTRALIA
COUNTRY OF AUTHOR: AUSTRALIA
SOURCE: INTERNATIONAL JOURNAL OF CLIMATOLOGY, (30 JUN 1997) Vol. 17, No. 8, pp. 831-845.
Publisher: JOHN WILEY & SONS LTD, BAFFINS LANE CHICHESTER, W SUSSEX, ENGLAND PO19 1UD.
ISSN: 0899-8418.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: PHYS
LANGUAGE: English
REFERENCE COUNT: 9

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI A climatology of coastal ridging over south-eastern Australia

AB The dominant feature of sea-level pressure (**SLP**) patterns over the coastal regions of south-eastern Australia is the ridging that extends northwards along the coast. This ridging is particularly evident in the warmer months, October to March, when it is present on part or all of approximately 65 per cent of days, usually occurring in sequences ranging from one to several days. In this paper, three distinct forms of coastal ridging are identified, and are referred to as types 1, 2 and 3. Their distinguishing synoptic and subsynoptic characteristics are described. As well as their **ubiquity** and synoptic significance, all three types are important because each can generate severe weather of the following types: heavy precipitation, strong winds, or sudden changes in wind direction and temperature. Climatologies of the three types are prepared for the 20-year period 1974-1993 in the form of both monthly and annual frequencies of occurrence. Given that south-eastern Australia is one of the areas of the globe most affected by the El Nino-Southern Oscillation (ENSO), correlations are calculated between monthly ridging

frequencies and monthly values of the Southern Oscillation Index (SOI). Also, annual frequencies are correlated with the annual (January to December) SOI value. Three diagnostic case studies are presented in considerable detail because they are very important to understand thoroughly the nature of and differences between the three types of ridging. (C) 1997 by the Royal Meteorological Society.

L11 ANSWER 61 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:80930 CAPLUS
DOCUMENT NUMBER: 126:167120
TITLE: Neither HMG-14a nor HMG-17 gene function is required for growth of chicken DT40 cells or maintenance of DNaseI-hypersensitive sites
AUTHOR(S): Li, Yi; Strahler, John R.; Dodgson, Jerry B.
CORPORATE SOURCE: Dep. of Microbiology, Michigan State Univ., East Lansing, MI, 48824, USA
SOURCE: Nucleic Acids Research (1997), 25(2), 283-288
CODEN: NARHAD; ISSN: 0305-1048
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Neither HMG-14a nor HMG-17 gene function is required for growth of chicken DT40 cells or maintenance of DNaseI-hypersensitive sites
AB HMG-14 and HMG-17 form a family of **ubiquitous** non-histone chromosomal proteins and have been reported to bind preferentially to regions of active chromatin structure. Our previous studies demonstrated that the chicken HMG-17 gene is dispensable for normal growth of the DT40 chicken lymphoid cell line. Here it is shown that the major chicken HMG-14 gene, HMG-14a, is also dispensable and, moreover, that DT40-derived cells lacking both HMG-17 and HMG-14a proteins show no obvious change in phenotype with respect to the parental DT40 cells. Furthermore, no compensatory changes in HMG-14b or histone protein levels were observed in cells lacking both HMG-14a and HMG-17, nor were any alterations detected in such hallmarks of chromatin structure as DNaseI-hypersensitive sites or micrococcal nuclease digestion patterns. It is concluded that the HMG-14a and HMG-17 proteins are not required for normal growth of avian cell lines in vitro, nor for the maintenance of DNaseI-hypersensitive sites in chromatin.

L11 ANSWER 62 OF 81 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 97:334346 SCISEARCH
THE GENUINE ARTICLE: BH61X
TITLE: Rapid X-ray variability in galactic black hole candidates
AUTHOR: vanderKlis M (Reprint)
CORPORATE SOURCE: UNIV AMSTERDAM, ASTRON INST ANTON PANNEKOEK, KRUISLAAN 403, NL-1098 SJ AMSTERDAM, NETHERLANDS (Reprint); NATL INST NUCL & HIGH ENERGY PHYS, CTR HIGH ENERGY ASTROPHYS, NL-1098 SJ AMSTERDAM, NETHERLANDS
COUNTRY OF AUTHOR: NETHERLANDS
SOURCE: ADVANCES IN SPACE RESEARCH, (10 MAY 1997) Vol. 19, No. 1, pp. 75-84.
Publisher: PERGAMON PRESS LTD, THE BOULEVARD LANGFORD LANE KIDLINGTON, OXFORD, ENGLAND OX5 1GB.
ISSN: 0273-1177.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 50
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
TI Rapid X-ray variability in galactic black hole candidates
AB The rapid X-ray variability of galactic accreting black-hole candidates is compared to that of accreting neutron stars with low magnetic fields. The power spectra of these objects can be described in terms of a small

number of simple power spectral shapes: power law noise, band limited noise, and quasi-periodic oscillations (QPO). In a given source, the properties of these power spectral components seem to depend, to first order, only on mass flux. Similarities in the power spectral properties between source types strongly suggest that similar physical mechanisms underlie power spectral components seen in black-hole candidates and in neutron stars with various magnetic-field strengths. Two rapid (greater than or similar to 1 Hz) QPO and two band limited noise components appear to occur across all types of X-ray binaries; the situation with respect to the **ubiquitous** power law components, as well as with respect to slow (less than or similar to 1 Hz) QPO is as yet unclear. One QPO and one band limited noise component appear to be magnetospheric as they are not seen in black-hole candidates and atoll sources (which are inferred to be neutron stars with a very low magnetic field). Another QPO and band limited noise component may be related to the presence of an inner, radiation pressure dominated accretion disk, as they do not occur in X-ray pulsars. It is discussed to what extent the relatively small quantitative differences between the rapid X-ray variability properties of neutron stars and black-hole candidates can be used to identify black holes, and whether there exist any qualitative differences (i.e., black hole signatures) in the rapid X-ray variability. (C) 1997 COSPAR.

L11 ANSWER 63 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:832474 CAPLUS
DOCUMENT NUMBER: 123:247892
TITLE: The chicken HMG-17 gene is dispensable for cell growth in vitro
AUTHOR(S): Li, Yi; Dodgson, Jerry B.
CORPORATE SOURCE: Dep. of Microbiology, Michigan State Univ., East Lansing, MI, 48824, USA
SOURCE: Molecular and Cellular Biology (1995), 15(10), 5516-23
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI The chicken HMG-17 gene is dispensable for cell growth in vitro
AB HMG-17 is a highly conserved and **ubiquitous** nonhistone chromosomal protein that binds to nucleosome core particles. HMG-17 and HMG-14 form a family of chromosomal proteins that have been reported to bind preferentially to regions of active chromatin structure. To study the functional role of the single-copy chicken HMG-17 gene, null mutants were generated by targeted gene disruption in a chicken lymphoid cell line, DT40. Heterozygous and homozygous null mutant cell lines were generated by two independent selection strategies. Heterozygous null mutant lines produced about half the normal level of HMG-17 protein, and homozygous null lines produced on detectable HMG-17. No significant changes in cell phenotype were observed in cells harboring either singly or doubly disrupted HMG-17 genes, and no compensatory changes in HMG-14 or histone protein levels were observed It is concluded that HMG-17 protein is not required for normal growth of avian cell lines in vitro, nor does the absence of HMG-17 protein lead to any major changes in cellular phenotype, at least in lymphoid cells.

L11 ANSWER 64 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 13
ACCESSION NUMBER: 1995:502815 CAPLUS
DOCUMENT NUMBER: 122:231114
TITLE: In vivo footprinting of an androgen-dependent enhancer reveals an accessory element integral to hormonal response
AUTHOR(S): Scarlett, Cameron O.; Robins, Diane M.
CORPORATE SOURCE: Dep. Human Genet., Univ. Michigan Med. Cent., Ann Arbor, MI, 48109-0618, USA
SOURCE: Molecular Endocrinology (1995), 9(4), 413-23

CODEN: MOENEN; ISSN: 0888-8809
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

TI In vivo footprinting of an androgen-dependent enhancer reveals an accessory element integral to hormonal response

AB A hormonally responsive enhancer that is specifically activated by androgens resides 2 kilobases upstream of the transcription start site of the mouse sex-limited protein (**Slp**) gene. The authors have previously shown that strong androgen induction in transfection requires a consensus hormone response element as well as several nonreceptor factor binding sites within this complex enhancer. To determine which accessory elements are required for androgen-dependent transcription, the authors have examined binding of nuclear proteins to the enhancer both in vitro and in vivo. In vitro footprinting assays demonstrated that multiple factors present in mouse liver and kidney nuclear exts. bound the enhancer, with tissue-specific but not sex-dependent differences in pattern. In contrast, examination of DNA sites occupied in liver chromatin identified a footprint (FPIV) that is well protected in males but sensitive to DNase I in females. FPIV was occupied in males in other sites of **Slp** expression, such as kidney, but not in tissues lacking expression, such as lung. FPIV protection was induced in females treated with androgen, abrogated in castrated males, and absent in immature mice, implying hormonal and developmental regulation of FPIV binding. Protection of the hormone response element, in contrast to FPIV, was not obvious but was discerned by anal. of densitometry data. Together with results from in vivo protein-DNA interactions determined for other steroid-dependent enhancers, this suggests that in some cases receptor may permit transcriptional activation by altering chromatin structure to allow access to other factors, which may not necessitate tight binding of receptor itself. This further emphasizes the crucial role of the nonreceptor factors in hormone response. The **ubiquitous** transcription factor Oct-1 forms complexes with an octamer motif present within FPIV by gel shift anal. with liver and kidney exts., making Oct-1 an intriguing candidate for partnership in androgen regulation.

L11 ANSWER 65 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 14
ACCESSION NUMBER: 1994:478821 CAPLUS
DOCUMENT NUMBER: 121:78821
TITLE: Functional redundancy: the respective roles of the two sloppy paired gene in Drosophila segmentation
AUTHOR(S): Cadigan, Kenneth M.; Grossniklaus, Ueli; Gehring, Walter
CORPORATE SOURCE: Biozentrum, University Basel, Basel, CH-4056, Switz.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1994), 91(14), 6324-8
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Functional redundancy: the respective roles of the two sloppy paired gene in Drosophila segmentation

AB The sloppy paired (**slp**) locus consists of 2 genes, slp1 and slp2, both of which encode proteins containing a forkhead domain (a DNA-binding motif). Previous work has shown that a severe segmentation phenotype is obtained only when both **slp** genes are deleted. Here the authors exam. the functional redundancy of the locus in more detail. The phenotypes of embryo containing various combinations of functional **slp** gene suggest that for early **slp** function, until gastrulation, only slp1 is required. At later times, there is still a greater requirement for slp1, but in many respects the 2 **slp** genes are completely redundant. Both **slp** genes produce similar phenotypes when **ubiquitously** expressed via a heat shock promoter. The authors propose that the **slp** proteins

are biochem. equivalent and that the greater requirement for slp1 in some functions can be explained in large part by its earlier expression.

L11 ANSWER 66 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 15
ACCESSION NUMBER: 1994:405468 CAPLUS
DOCUMENT NUMBER: 121:5468
TITLE: Localized expression of sloppy paired protein maintains the polarity of Drosophila parasegments
AUTHOR(S): Cadigan, Kenneth M.; Grossniklaus, Ueli; Gehring, Walter J.
CORPORATE SOURCE: Biozentrum, Univ. Basel, Basel, CH-4056, Switz.
SOURCE: Genes & Development (1994), 8(8), 899-913
CODEN: GEDEEP; ISSN: 0890-9369
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Localized expression of sloppy paired protein maintains the polarity of Drosophila parasegments
AB During germ-band extension in the Drosophila embryo, intercellular communication is required to maintain gene expression patterns initiated at cellular blastoderm. For example, the wingless (wg) single-cell-wide stripe in each parasegment (PS) is dependent on a signal from the adjacent, posterior cells, which express engrailed (en). This signal is thought to be the hedgehog (hh) gene product, which antagonizes the activity of patched (ptc), a repressor of wg expression. Genetic evidence indicates that the hh signal is bidirectional, but wg transcription is only derepressed on the anterior side of the en/hh stripes. To explain the asym. response of the wg promoter to the hh signal, current models predict that each PS is divided into cells that are competent to express either wg or en, but not both. The sloppy paired (**slp**) locus contains 2 transcription units, both encoding proteins containing a forkhead domain, a DNA-binding motif. Removal of **slp** gene function causes embryos to exhibit a severe pair-rule/segment polarity phenotype. The en stripes expand anteriorly in **slp** mutant embryos and **slp** activity is an absolute requirement for maintenance of wg expression at the same time that wg transcription is dependent on hh. The **slp** proteins are expressed in broad stripes just anterior of the en-pos. cells, overlapping the narrow wg stripes. It is proposed that by virtue of their ability to activate wg and repress en expression, the distribution of the **slp** proteins define the wg-competent and en-competent groups. Consistent with this hypothesis, **ubiquitous** expression of **slp** protein throughout the PS abolishes en expression and, in ptc mutant embryos, results in a near **ubiquitous** distribution of wg transcripts. In addition to demonstrating the role of **slp** in maintaining segment polarity, the results suggest that **slp** works in, or parallel with, the ptc/hh signal transduction pathway to regulate wg transcription.

L11 ANSWER 67 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 16
ACCESSION NUMBER: 1993:248071 CAPLUS
DOCUMENT NUMBER: 118:248071
TITLE: Male-specific expression of mouse sex-limited protein requires growth hormone, not testosterone
AUTHOR(S): Georgatsou, Elena; Bourgarel, Pierre; Meo, Tommaso
CORPORATE SOURCE: Unite Immunogenet., Inst. Natl. Sante Rech. Med., Paris, 75724, Fr.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1993), 90(8), 3626-30
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Male-specific expression of mouse sex-limited protein requires growth hormone, not testosterone
AB Sex-limited protein (**Slp**), an isoform of mouse complement

component C4, is expressed predominantly in liver and nearly exclusively in sexually mature males or testosterone-treated females. It is demonstrated that the pronounced rise of C4-**Slp** mRNA promoted by androgens in the liver is due to nuclear factors acting at a transcriptional stage. Hypophysectomized animals of either sex fail to express the C4-**Slp** gene and are refractory to testosterone. However, gene expression at male levels is restored even more promptly by injections of growth hormone alone. Addnl., animals carrying an **ubiquitously** expressed human growth hormone transgene lack C4-**Slp** mRNA and are insensitive to testosterone treatment. That growth hormone is sufficient to induce expression in a manner independent of androgen-receptor activity is shown by the hormonal treatment of Tfm mice. These androgen receptor-defective animals lack C4-**Slp** mRNA, which however can be fully induced by growth hormone injections. Apparently, the sexual dimorphism of C4-**Slp** expression employs liver nuclear mediators distinct from those directly instructed by androgens and is brought about by the intermittent rise of growth hormone, dictated by testosterone.

L11 ANSWER 68 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:401096 CAPLUS
DOCUMENT NUMBER: 117:1096
TITLE: Detection of thymosin β4 in situ in a guinea pig cerebral cortex preparation using proton NMR spectroscopy
AUTHOR(S): Kauppinen, Risto A.; Nissinen, Terhi; Karkkainen, Anne Mari; Pirttila, Tiina R. M.; Palvimo, Jorma; Kokko, Harri; Williams, Stephen R.
CORPORATE SOURCE: Dep. Biochem. Biotechnol., Univ. Kuopio, Kuopio, Finland
SOURCE: Journal of Biological Chemistry (1992), 267(14), 9905-10
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Detection of thymosin β4 in situ in a guinea pig cerebral cortex preparation using proton NMR spectroscopy
AB The macromols. that contribute to the brain 1H NMR spectrum were investigated. The cerebral cortex showed distinct resonance at the uncrowded methyl- and methylene chemical shift scale of the spin-echo 1H NMR spectrum. The peaks at 1.22 and 1.40 ppm (relative to the Me protons of N-acetyl aspartate at 2.02 ppm) arise from cerebral macromols. without evidence for coresonances from low-mol.-weight metabolites as shown by the spin-spin relaxation decays of these resonances. In addition to these NMR signals, peaks at 0.9 and 1.7 ppm from macromols. were detected. These resonances are from proteins, and the polypeptides that contributed to the 1H NMR peaks were identified. Two proteins that were present at concns. of 250 and 350 μg/g of dried tissue showed 1H NMR spectra that resembled the macromol. pattern in the cerebral 1H NMR spectrum. They were identified as thymosin β4 and histone H1, resp. Thymosin β4 was present in the soluble high-speed cytoplasmic fraction and in the P2 pellet, whereas histone H1 was detected in the nuclear-enriched fraction. A chemical shift-correlated 2-dimensional 1H NMR spectrum of thymosin β4 in vitro revealed a coupling pattern that matched the previously noted macromol. in the cerebral cortex. On the basis of both 1- and 2-dimensional NMR evidence, subcellular distribution, and high concentration, the 1H NMR signals at 0.9, 1.22, 1.40, and 1.7 ppm in the cerebral cortex were assigned to thymosin β4.

L11 ANSWER 69 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:626736 CAPLUS
DOCUMENT NUMBER: 117:226736

TITLE: The dynamic properties of neuronal chromatin are modulated by triiodothyronine
AUTHOR(S): Cestelli, Allesandro; Gristina, Roberto; Castiglia, Daniele; Di Liegro, Carlo; Savettieri, Giovanni; Salemi, Guiseppe; Di Liegro, Italia
CORPORATE SOURCE: Dip. Biol. Cell Sviluppo "Alberto Monroy", Palermo, 90123, Italy
SOURCE: Neurochemical Research (1992), 17(11), 1049-55
CODEN: NEREDZ; ISSN: 0364-3190
DOCUMENT TYPE: Journal
LANGUAGE: English

TI The dynamic properties of neuronal chromatin are modulated by triiodothyronine

AB The effect of T3 on the rate of synthesis of nuclear proteins was studied during terminal differentiation of rat cortical neurons cultured in a serum-free medium. To this aim total and acid-soluble nuclear proteins were analyzed by different electrophoretic techniques. During maturation in vitro, neuronal nuclei undergo a dramatic change in the rate at which different classes of histones and high mobility group (HMG) proteins are synthesized. The synthetic activity, measured as incorporation of radioactive precursors into nuclear proteins, slows down with age. Especially evident is the decrease in core histones synthesis; at day 15, on the other hand, HMG 14 and 17 and **ubiquitinated** H2A (A24) are synthesized at a high rate, especially in T3-treated neurons. Neurons treated with T3 show, at any age tested, a higher level of lysine incorporation into nuclear proteins. Even if during the first days of culture neurons synthesize core histones more actively in the presence of T3, there is no accumulation of these proteins at later stages, as compared with untreated cells. Possible implications of these data and relations with the chromatin rearrangement which accompanies neuronal terminal differentiation are discussed.

L11 ANSWER 70 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:546488 CAPLUS
DOCUMENT NUMBER: 117:146488
TITLE: Contribution of cytoplasmic polypeptides to the proton NMR spectrum of developing rat cerebral cortex
AUTHOR(S): Kauppinen, Risto A.; Palvimo, Jorma
CORPORATE SOURCE: Dep. Biochem. Biotechnol., Univ. Kuopio, Kuopio, Finland
SOURCE: Magnetic Resonance in Medicine (1992), 25(2), 398-407
CODEN: MRMEEN; ISSN: 0740-3194
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Contribution of cytoplasmic polypeptides to the proton NMR spectrum of developing rat cerebral cortex

AB The contribution of cytoplasmic peptides thymosins β4 and β10 to the 1H NMR spectrum during the maturation of the rat cerebral cortex was assessed. In the proton spectrum intense broad peaks at 0.9, 1.22, and 1.40 ppm from thymosins decreased in size relative to the signal at 2.02 ppm in parallel to the reciprocal increase in the concentration of N-acetylaspartate. Levels of thymosins β4 and β10 were under developmental regulation. It is concluded that peaks from thymosins may provide extended information for an NMR spectroscopist, and thus they have to be taken into account in the interpretation of the newborn cerebral 1H NMR spectrum.

L11 ANSWER 71 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:33652 CAPLUS
DOCUMENT NUMBER: 118:33652
TITLE: Large scale cDNA sequencing for analysis of quantitative and qualitative aspects of gene expression

AUTHOR(S): Okubo, Kousaku; Hori, Naohiro; Matoba, Ryo; Niiyama, Toshiyuki; Fukushima, Atsushi; Kojima, Yuko; Matsubara, Kenichi
CORPORATE SOURCE: Inst. Mol. Cell. Biol., Osaka Univ., Suita, 565, Japan
SOURCE: Nature Genetics (1992), 2(3), 173-9
CODEN: NGENEC; ISSN: 1061-4036
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Large scale cDNA sequencing for analysis of quantitative and qualitative aspects of gene expression

AB Large scale sequencing of cDNAs provides a complementary approach to structural anal. of the human genome by generating expressed sequence tags (ESTs). Large-scale sequencing was undertaken of a 3'-directed cDNA library from the human liver cell line HepG2, that is a non-biased representation of the mRNA population. 982 Random cDNA clones were sequenced yielding more than 270 kilobases. A significant portion of the identified genes encoded secretable proteins and components for protein-synthesis. The abundance of cDNA species varied from 2.2% to <0.004%. Fifty-two percent of the mRNA were abundant species consisting of 173 genes and the rest were non-abundant, consisting of .apprx.6600 genes.

L11 ANSWER 72 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:211680 CAPLUS
DOCUMENT NUMBER: 116:211680
TITLE: Qualitative differences in nuclear proteins correlate with neuronal terminal differentiation
AUTHOR(S): Cestelli, Alessandro; Castiglia, Daniele; Di Liegro, Carlo; Di Liegro, Italia
CORPORATE SOURCE: Dip. Biol. Cell. Sviluppo "Alberto Monroy", Univ. Palermo, Palermo, 90123, Italy
SOURCE: Cellular and Molecular Neurobiology (1992), 12(1), 33-43
CODEN: CMNEDI; ISSN: 0272-4340
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Qualitative differences in nuclear proteins correlate with neuronal terminal differentiation

AB The protein composition of neuronal nuclei was studied at two stages of brain maturation, i.e, before (embryonic day 616) and after (postnatal day 10) shortening of the nucleosome repeat length. Glial nuclei were analyzed in parallel as a control. Total nuclear or HCl- and 5% perchloric acid-soluble proteins were analyzed by different electrophoretic techniques. The results show an increase in the concentration of histone H1° with differentiation, although the H1 class undergoes an overall decrease. The chromatin of mature neurons is also enriched in the ubiquinated form of histone H2A (A24), while the high-mobility group (HMG) proteins 1 and 2 seem to decrease slightly relative to core histones. Both quant. and qual. differences in the abundance of nonhistone proteins relative to histones accompany neuronal terminal differentiation.

L11 ANSWER 73 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1991:600068 CAPLUS
DOCUMENT NUMBER: 115:200068
TITLE: Assessment of the transcriptional activation potential of the HMG chromosomal proteins
AUTHOR(S): Landsman, David; Bustin, Michael
CORPORATE SOURCE: Natl. Cent. Biotechnol. Inf., Natl. Libr. Med., Bethesda, MD, 20894, USA
SOURCE: Molecular and Cellular Biology (1991), 11(9), 4483-9
CODEN: MCEBD4; ISSN: 0270-7306
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Assessment of the transcriptional activation potential of the HMG chromosomal proteins

AB Chromosomal proteins HMG-14, HMG-17, and HMG-1 are among the most abundant, **ubiquitous**, and evolutionarily conserved nonhistone proteins. Anal. of their structure reveals features which are similar to those of certain transcription factors. The distribution of charged amino acid residues along the polypeptide chains is asym.: pos. charges are clustered toward the N-terminal region, while neg. charges are clustered toward the C-terminal region. The residues in the C-terminal region have the potential to form α helices with neg. charged surfaces. The abilities of HMG-14, -17, and -1 to function as transcriptional activators were studied in Saccharomyces cerevisiae cells expressing LexA-HMG fusion proteins (human HMG-14 and -17 and rat HMG-1) which bind to reporter mols. containing the β-galactosidase gene downstream from a lexA operator with in vitro-synthesized fusion proteins shows that there are more sites for HMG-14, -17, and -1 binding than for LexA binding and that only the fusion constructs which contain the C-terminal, acidic domains of HMG-17 bind the lexA operator specifically. None of the LexA-HMG fusion protein constructs elevate the level of β-galactosidase activity in transfected yeast cells. Thus, although HMG-14, -17, and -1 are structurally similar to acidic transcriptional activators, these chromosomal proteins do not function as activators in this test system.

L11 ANSWER 74 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1988:201079 CAPLUS
DOCUMENT NUMBER: 108:201079
TITLE: Separation and quantification of histone H1 subtypes and high-mobility-group proteins by reversed-phase liquid chromatography: protein levels in rat tissues during postnatal development
AUTHOR(S): Karhu, Inkeri; Mahonen, Anitta; Palvimo, Jorma
CORPORATE SOURCE: Dep. Biochem., Univ. Kuopio, Kuopio, SF-70211, Finland
SOURCE: Journal of Chromatography (1988), 426(1), 65-73
CODEN: JOCRAM; ISSN: 0021-9673
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Separation and quantification of histone H1 subtypes and high-mobility-group proteins by reversed-phase liquid chromatography: protein levels in rat tissues during postnatal development

AB The rapid separation and quantification of histone H1 subtypes and high-mobility-group (HMG) chromatin proteins by reversed-phase liquid chromatog. on a butylsilica-based column is described. The proteins were fractionated by means of a multistep MeCN gradient containing 0.1% trifluoroacetic acid. This system is capable of resolving the 4 main HMG proteins (1, 2, 14, and 17), HMG I, protein P1 with HMG 18 and HMG 19 (in 1 peak), and 5 histone H1 subtypes in a single 33-min anal. This method was used to study levels of these chromosomal proteins in nuclei of rat liver, spleen, testis, and thymus during postnatal development during 1-20 wk of age. Although no clear tissue specificity of the HMG proteins was apparent, there was significant differences in the relative amts. of these proteins in different tissues. The relative amount of HMG 1 increased at 1-12 wk of age and decreased thereafter, whereas those of HMG 14 and HMG 17 remained almost unchanged. Marked quant. differences were observed in the 5 histone H1 subtypes in different tissues. The largest changes in their levels during development were found in the liver and the smallest changes in the thymus. The changes in the spleen and testis were intermediate. These results suggest that the changes in the relative amts. of histone H1 subtypes and HMG proteins observed during postnatal development of the rat may result from differences in the structure of chromatin in these tissues and these tissues and thus reflect the activity of mol. mechanisms involved in replication and differentiation of the cells.

L11 ANSWER 75 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1989:434464 CAPLUS
DOCUMENT NUMBER: 111:34464
TITLE: Probing MHC DNA in search of coding sequences: detection of a novel gene located between C4 and Bf in mouse and man
AUTHOR(S): Levi-Strauss, Matthieu; Carroll, Michael; Steinmetz, Michael; Meo, Tommaso
CORPORATE SOURCE: Inst. Pasteur, Paris, 75724/15, Fr.
SOURCE: NATO ASI Series, Series A: Life Sciences (1987), 144(H-2 Antigens: Genes, Mol., Funct.), 305-14
CODEN: NALSDJ; ISSN: 0258-1213
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Probing MHC DNA in search of coding sequences: detection of a novel gene located between C4 and Bf in mouse and man

AB The localization of the murine gene corresponding to the WL623 cDNA clone was confirmed and its transcriptional orientation was determined using cosmids from another genomic library (B10.W7R, H-2w7). In this halotype, the gene corresponding to the WL623 cDNA clone is situated between the Bf gene and one of the four C4-**Slp** gene copies characteristically amplified in the strain. The 3' end of the gene was found to be very close to the Bf gene, whereas its 5' end is some 20 kb away from C4-**Slp**. Surprisingly, this new gene has a transcriptional orientation unlike the other class III genes. In man, the gene is also located in the corresponding genetic region. Indeed, the WL623 cDNA clone, when used as a probe on Southern blots of human DNA, detects a unique sequence in the genome present in the human cosmid clone 1a covering the region situated between the C4A and Bf genes in the human major histocompatibility complex. Thus, a new gene, expressed as an 18 S polyadenylated RNA, transcribed **ubiquitously** and located in H-2 and HLA, was found between the C4 and Bf genes. The sequence derived from liver cDNA clones indicates that this new gene may encode a 42 kDa polypeptide.

L11 ANSWER 76 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1986:437753 CAPLUS
DOCUMENT NUMBER: 105:37753
TITLE: The active immunoglobulin κ chain gene is packaged by non-**ubiquitin**-conjugated nucleosomes
AUTHOR(S): Huang, Sue Ying; Barnard, Mary B.; Xu, Ming; Matsui, Seiichi; Rose, Stephen M.; Garrard, William T.
CORPORATE SOURCE: Health Sci. Cent., Univ. Texas, Dallas, TX, 75235, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1986), 83(11), 3738-42
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English

TI The active immunoglobulin κ chain gene is packaged by non-**ubiquitin**-conjugated nucleosomes

AB The mol. features of active chromatin were studied by mapping, by 2-dimensional electrophoresis, the protein composition of nucleosomes that package the Ig κ chain gene of mouse plasmacytoma cells. Nucleoprotein particles that possess the active κ chain gene comigrate with bulk mononucleosomes that contain high-mobility-group proteins HMG-14 or -17 but lack histone H1. High electrophoretic resolution of the underlying core particles, after removal of **ubiquitin** by isopeptidase treatment, reveals that these nucleosomes are nonubiquitinated, even though they coincidently migrate with bulk **ubiquitinated** particles. This distinctive electrophoretic behavior may be correlated with the presence of histone H2A.X. Nucleosomes exhibiting these unusual properties appear to span ≥10 kilobases in both transcribed and nontranscribed regions, suggesting that mechanisms independent of transcription exist to initiate, maintain, and

propagate a common chromatin phenotype over long distances along the κ chain locus.

L11 ANSWER 77 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1983:534738 CAPLUS
DOCUMENT NUMBER: 99:134738
TITLE: Cellular and SV40 chromatin: replication, segregation, **ubiquitination**, nuclease-hypersensitive sites, HMG-containing nucleosomes, and heterochromatin-specific protein
AUTHOR(S): Varshavsky, A.; Levinger, L.; Sundin, O.; Barsoum, J.; Ozkaynak, E.; Swerdlow, Paul S.; Finley, D.
CORPORATE SOURCE: Dep. Biol., Massachusetts Inst. Technol., Cambridge, MA, 02139, USA
SOURCE: Cold Spring Harbor Symposia on Quantitative Biology (1983), Volume Date 1982, 47(1), 511-28
CODEN: CSHSAZ; ISSN: 0091-7451
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Cellular and SV40 chromatin: replication, segregation, **ubiquitination**, nuclease-hypersensitive sites, HMG-containing nucleosomes, and heterochromatin-specific protein
AB A discussion is presented of published and unpublished studies concerning replicative intermediates of SV40 virus, mechanisms for the termination of DNA synthesis by SV40 and the segregation of daughter SV40 chromosomes, the enzymic probing of the origin-containing exposed region of SV40 chromosomes, the distribution of **ubiquitinated** and other variant nucleosomes from Drosophila melanogaster, the distribution of **ubiquitinated** nucleosomes and high-mobility-group (HMG) proteins within the mouse dihydrofolate reductase [9002-03-3] gene, and on the mechanism of preferential in vitro binding of proteins HMG14 and **HMG17** to nucleosomes derived from transcribed genes.

L11 ANSWER 78 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 17
ACCESSION NUMBER: 1983:85138 CAPLUS
DOCUMENT NUMBER: 98:85138
TITLE: Affinity of **HMG17** for a mononucleosome is not influenced by the presence of **ubiquitin**-H2A semihistone but strongly depends on DNA fragment size
AUTHOR(S): Swerdlow, Paul S.; Varshavsky, Alexander
CORPORATE SOURCE: Dep. Biol., Massachusetts Inst. Technol., Cambridge, MA, 02139, USA
SOURCE: Nucleic Acids Research (1983), 11(2), 387-401
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Affinity of **HMG17** for a mononucleosome is not influenced by the presence of **ubiquitin**-H2A semihistone but strongly depends on DNA fragment size
AB A 2-dimensional electrophoretic binding assay was used to study the interaction of purified high-mobility group protein 17 (**HMG17**) with isolated HeLa mononucleosomes as a function of DNA fragment size and the presence of **ubiquitin**-H2A semihistone. No significant difference in the affinities of **HMG17** for core mononucleosomes in the presence or absence of **ubiquitin** was observed In striking contrast, the apparent affinity of **HMG17** for mononucleosomes increases >100-fold on increasing the length of the mononucleosomal DNA fragment by as few as 3-5 base pairs (bp) over the core DNA length (.apprx.146 bp). The magnitude of this effect may be sufficient to explain the preferential binding of **HMG17** in vitro to mononucleosomes derived from actively transcribed genes.

L11 ANSWER 79 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1982:176851 CAPLUS
DOCUMENT NUMBER: 96:176851
TITLE: Effect of high mobility group nonhistone proteins HMG-20 (**ubiquitin**) and HMG-17 on histone deacetylase activity assayed in vitro
AUTHOR(S): Mezquita, J.; Chiva, M.; Vidal, S.; Mezquita, C.
CORPORATE SOURCE: Fac. Med., Univ. Barcelona, Barcelona, Spain
SOURCE: Nucleic Acids Research (1982), 10(5), 1781-97
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Effect of high mobility group nonhistone proteins HMG-20 (**ubiquitin**) and HMG-17 on histone deacetylase activity assayed in vitro
AB A method previously described by R. Reeves and E. P. M. Candido (1980) was used to partially release histone deacetylase from cell nuclei together with putative regulators of the enzyme. Histone deacetylase released from testis cell nuclei and its putative regulators were separated by gel filtration on Sepharose 6B. A peak of low mol. weight contains a heat-stable factor that stimulates histone deacetylase in vitro. Many of the properties of the activator coincide with those of the protein HMG-20 (**ubiquitin**). **Ubiquitin** isolated from testis cell nuclei stimulated histone deacetylase in vitro. It has been suggested that HMG-17 partially inhibits histone deacetylase in Friend cell nuclei. In the testis system, HMG-17 shows no inhibitory effect on histone deacetylase activity.

L11 ANSWER 80 OF 81 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1981:437714 CAPLUS
DOCUMENT NUMBER: 95:37714
TITLE: Structure of nucleosome core particles containing uH2A (A24)
AUTHOR(S): Kleinschmidt, Ann M.; Martinson, Harold G.
CORPORATE SOURCE: Mol. Biol. Inst., Univ. California, Los Angeles, CA, 90024, USA
SOURCE: Nucleic Acids Research (1981), 9(11), 2423-31
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Structure of nucleosome core particles containing uH2A (A24)
AB Histone uH2A (A24), a modification of histone H2A involving the covalent attachment of **ubiquitin**, was purified and reconstituted, in place of H2A, into high-mol.-weight nucleohistone containing core histones and DNA. Histone uH2A-containing core particles were then prepared by nuclease digestion. Two uH2A mols. can be accommodated within a core particle, and the presence of these 2 uH2A mols. in a core particle does not significantly alter either the pattern or the rate of DNase I digestion as compared to both reconstituted and native core particles. HMG proteins 14 and 17 bound to uH2A-containing core particles. Thus, uH2A has little influence on structure at the level of individual nucleosomes.

L11 ANSWER 81 OF 81 MEDLINE on STN
ACCESSION NUMBER: 82095171 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6172276
TITLE: Characterisation of a chromatin fraction bearing pulse-labelled RNA. 2. Quantification of histones and high-mobility-group proteins.
AUTHOR: Gabrielli F; Hancock R; Faber A J
SOURCE: European journal of biochemistry / FEBS, (1981 Nov) 120 (2) 363-9.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198203
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19820322

TI Characterisation of a chromatin fraction bearing pulse-labelled RNA. 2. Quantification of histones and high-mobility-group proteins.

AB The histone variants and high-mobility-group (HMG) proteins of a transcribing fraction of chromatin, described in the preceding paper of this journal, have been analysed qualitatively and quantitatively by a combination of one-dimensional and two-dimensional gel electrophoresis. The stoichiometry of the four core histones (all variants included) in this fraction is equimolar and is not detectably different from that in the nontranscribing fraction or in total chromatin. The molar ratio of histone H1 to the core histones is markedly lower, by approximately 72%, than that in the nontranscribing fraction. A minor histone variant identified as M1 (an H2A variant) is detected only in the transcribing fraction, while variant H3.1 is found only in the non-transcribing fraction. Proteins A24, HMG1 and HMG2 are essentially absent from the transcribing fraction; HMG14 is found uniquely in this fraction, while **HMG17** occurs at a relatively lower level.

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(FILE 'HOME' ENTERED AT 17:36:13 ON 30 MAY 2005)

FILE 'REGISTRY' ENTERED AT 17:36:25 ON 30 MAY 2005
E APRATAXIN/CN
L1 1 S E4
L2 8 S E5-E12

FILE 'CAPLUS, BIOSIS, SCISEARCH, MEDLINE' ENTERED AT 17:38:58 ON 30 MAY 2005

FILE 'REGISTRY' ENTERED AT 17:39:16 ON 30 MAY 2005
SET SMARTSELECT ON
L3 SEL L1 1- CHEM : 4 TERMS
SET SMARTSELECT OFF

FILE 'CAPLUS, BIOSIS, SCISEARCH, MEDLINE' ENTERED AT 17:39:16 ON 30 MAY 2005
L4 1 S L3

FILE 'REGISTRY' ENTERED AT 17:39:53 ON 30 MAY 2005
SET SMARTSELECT ON
L5 SEL L2 1- CHEM : 28 TERMS
SET SMARTSELECT OFF

FILE 'CAPLUS, BIOSIS, SCISEARCH, MEDLINE' ENTERED AT 17:39:54 ON 30 MAY 2005
L6 4 S L5
L7 0 S UBIQUIT? AND (L4 OR L6)
L8 137114 S UBIQUIT?
L9 7319 S (SLP OR HMG17 OR PINX1 OR CIR OR HMGN3 OR HSPC144)
L10 120 S L8 AND L9
L11 81 DUP REM L10 (39 DUPLICATES REMOVED)

=> log off
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

STN INTERNATIONAL LOGOFF AT 17:47:26 ON 30 MAY 2005